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ALZHEIMER'S DISEASE WITH PREDOMINATING CROSSED CEREBRO-CEREBELLAR HEMIATROPHY

BY

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(Received 18th February, 1941)

IN spite of the great number of contributions on the subject of Alzheimer's disease and its position amongst the presenile cerebral degenerations the character of the disorder is still obscure. We have investigated a case with uncommon features which we believe throws fresh light on old problems and in itself suggests new lines of investigation.

Annie O., was admitted to the Bristol Mental Hospital on 21st September, 1934, at the age of 53. Her history was as follows. No history of insanity or of specific physical illness in the family. Parents and grand-parents exceptionally long-lived. The patient herself was an intelligent child who did very well at school. She showed no noticeable mental or physical abnormality until after the birth of her only child when she was 41. The confinement was normal, but some time after the patient found difficulty in looking after the house and child. During the next eight years her intellectual powers appear to have deteriorated slowly so that she became unable to attend to her shopping or to be trusted alone in her house. She used to wander from her house and forget where she lived and did not know the date or the value of money. At the time of the menopause at 49 her mental symptoms appeared to have become more pronounced; four years later, on 21st September, 1934, she entered the mental hospital.

On admission she presented a picture of depression. She said that she felt miserable and was unable to do her work. She stated that she felt queer at times and that she was afraid of falling. The patient was quiet and well behaved and had sufficient comprehension of her mental disability to seek treatment of her own accord. There seemed to be no error of perception. Her memory was very poor and she could not even remember how many children she had. Actually she had only one.

She did not know the date and had no knowledge at all of recent events. It was noted that she would forget the position of her bed in the ward.

Physical State.—The patient was rather thin but apart from a slight trace of albumen in the urine there was no sign of physical illness. The hair was grey, the complexion sallow, and there was a commencing arcus senilis. No sign of disorder of the C.N.S. Wassermann reaction in blood negative.

Course of Illness.—During the first year of hospitalization the patient's mental condition underwent a slow deterioration. Her memory became if anything worse and while at first she could do simple tasks, such as elementary sewing, by the end of 12 months she was unable to dress and wash herself unaided. She had, however, ceased to be depressed, she looked cheerful, and always expressed the belief that she was getting better. During the next four years the deterioration continued and the case history suggests that by that time she showed aphasic phenomena. She paid little attention to what was said to her and when asked questions she merely repeated her own name. It was difficult to examine her as she was very unco-operative. She sat continually in dark corners with head bent wringing her hands. She was considerably wasted. Within a year before her death she became greatly emaciated; she was feeble and incontinent requiring constant nursing attention. She had no fits in the course of the illness. She died on 11th February, 1940, of broncho-pneumonia.

Case Summary.—This is a case of a patient in whom intellectual deterioration made its appearance at the age of 41. This deterioration was slowly progressive until the death of the patient at 59. The symptoms were those of an organic dementia, failure of memory being the earliest and most prominent symptom. Towards the end the patient was unable to look after herself. In the late stages of the illness the function of speech was disturbed and the case history suggests that this disturbance was chiefly of the executive ability. Death was due to pneumonia. During the whole course of the illness there was no evidence of delusions, hallucinations or fits, nor were there signs of paralysis. There were neither signs nor history of chronic alcoholism nor any evidence of severe cardiovascular or renal disorder.

Post-mortem Examination.—Apart from brain, the only significant findings were : Generalized wasting of body. Arteries soft. Slight atheroma of ascending aorta. Heart small and firm, no valvular nor endocardial abnormality. Lungs : Typical picture, confirmed microscopically, of a confluent broncho-pneumonia. Kidneys : Early fibrosis. No noteworthy abnormalities in other organs.

Brain.—Macroscopical Findings : Dura normal, pia-arachnoid moderately thickened and opaque, freely separable from the brain surface. Cerebro-spinal fluid present in marked quantity in the subarachnoid space. Weight without dura 870 grm. There was gross generalized asymmetrical atrophy involving both hemispheres of the brain and of the cerebellum. The right cerebral hemisphere was noticeably much more wasted than the left, the contralateral left cerebellar hemisphere being more wasted than the other. The difference in the degree of atrophy between the cerebral hemispheres was proportionately the same as between those of the cerebellum. The degree of atrophy was not uniform throughout all regions of the cerebral cortex of each side. The order of maximum wasting was the same on each side, namely, frontal, occipital, temporal, parietal lobes. The anterior and posterior central gyri were less affected. The degree of atrophy of the various lobes of the cerebellum was uniform, apart from the asymmetry. The great disparity in the amount of the atrophic process is shown in the photograph taken from above (Fig. 1). The marked pro-

jection of the right cerebellar hemisphere behind the right occipital pole is strikingly demonstrated, in contrast to the normal relationship of occipital lobe and cerebellum when viewed from above. There was diffuse gross atrophy of both cerebellar hemispheres but, as mentioned before, that of the left side was the more extreme. This is illustrated in Fig. 2 which shows from the postero-caudal aspect the asymmetry of the two cerebellar hemispheres and the abnormal relationship between the left occipital pole and the left cerebellar hemisphere. The individual cerebellar folia of the left side appeared more atrophic so that there was a considerable difference in the size of the corresponding parts of the cerebellar hemisphere of each side.

Serial sections in the frontal plane revealed a diffuse hydrocephalus affecting all ventricles, those of the right cerebral hemisphere being more extensively enlarged.

The pituitary showed no significant macroscopic features.

Microscopical Findings.—Diffuse increase of fibrous tissue in the leptomeninges without signs of inflammation.

Cerebral Cortex.—Nissl staining revealed generalized reduction in the number of cells according to the degree of gross atrophy of the area concerned. The loss of cells was most pronounced in the 3rd and 5th layer of the cortex. There was an increased accumulation of lipofuscin, especially in the pyramidal elements many of which cells were disorientated. There were many small areas of " dropping out " of ganglion cells. All these abnormalities, corresponding to what is generally described as senile changes of ganglion cells, were most pronounced in the frontal and occipital lobes and in the cornu Ammonis of both sides, but more especially of the right. There were similar degenerative changes in the basal ganglia and in the inferior olives.

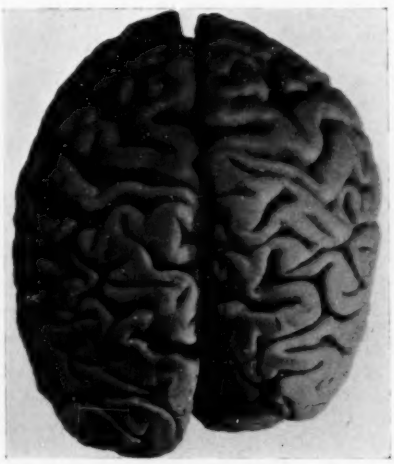

Fig. 1.—Brain viewed from above, showing predominatingly wasted right cerebral hemisphere and the projecting right cerebellar hemisphere.

Fig. 2.—Brain viewed from the postero-caudal aspect showing increased relative wasting of the left cerebellum (right in the photograph) and right cerebrum.

v. Braunmühl's silver stain revealed an enormous number of senile plaques of all known sizes and types (Figs. 3, 4, 5). There were large coarse plaques with a central clear space with or without nucleus, plaques with a darkly stained nucleus and a loosely arranged outer zone, irregular patches of argentophile substance without formation of nuclei, some of them surrounding blood vessels. The plaques were seen in all layers of the cortex, being most numerous in the 3rd and 5th layer, of sparsest distribution and of the smallest calibre in the 1st. Their number was relatively greater in the right hemisphere and in the frontal and occipital lobes and in

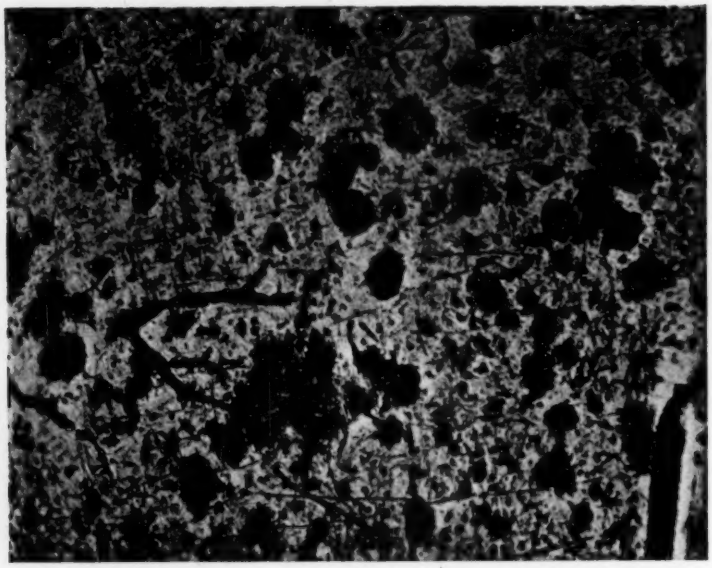

Fig. 3.—Right frontal cortex showing numerous argentophile plaques of various types and pyramidal cells with neurofibrillary change. Some of the cells are disorientated. v. Braunmühl's silver impregnation.

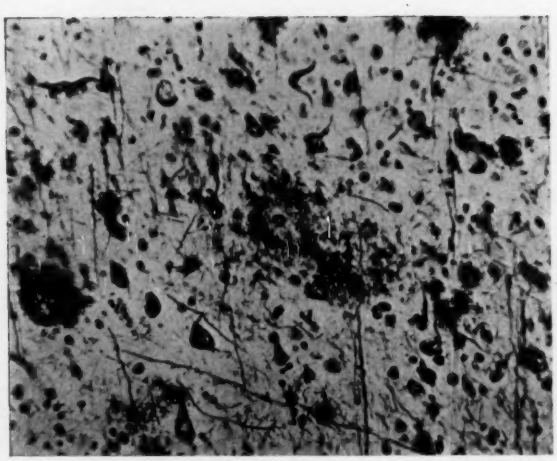

Fig. 4.—Right frontal cortex showing numerous pyramidal cells with advanced neurofibrillary change and plaques. v. Braunmühl's silver impregnation.

Fig. 5.—Right frontal cortex showing numerous excrescences on the axis cylinders (" knotted canes "). v. Braunmühl's silver impregnation.

the cornua Ammonis. Numerous smaller plaques were found in the striate body and in the pallidum. There were single elliptic plaques in the white matter adjoining the cortex. The degree of cell changes described above as well as the number of the plaques was least in the cortex of the anterior and posterior central gyri.

Alzheimer's neurofibrillary change of the ganglion cells was encountered all over the cortex, most pronounced in degree and distribution in the frontal and occipital areas and in the cornua Ammonis where it was seen in nearly all pyramidal cells, least in the gyri centrales. Fig. 4 demonstrates these changes affecting mainly the pyramidal cells. The large cells of Beetz in the 5th layer of the anterior central gyrus were affected to a relatively slight extent.

There were marked and unusual changes of the axis cylinders ; many were swollen, the argentophile substance showing signs of granular disintegration. Most impressive, however, was the presence of numerous excrescences of the shape of knots and buds which gave the axis cylinder in longitudinal aspect the appearance of a knotted cane or walking stick (Fig. 5). Higher magnification revealed that these excrescences consisted of a homogeneous argentophile substance. The distribution of these changes corresponded with that of the plaques. They were found in small numbers in the basal ganglia and in the cerebellar cortex.

Myelin staining showed typical degenerative changes in all cortical layers and in the basal ganglia. The myelin sheaths were swollen, there were myelin balls in many places. A certain amount of diffuse demyelinization appeared to have taken place in the most atrophic areas of the cortex.

The changes of the neuroglial elements were typical, according to what has been described in similar cases. There was intense gliosis, especially around the senile plaques and affecting each of the glial elements which were enormously hypertrophied and increased in number. In many places the cerebral histiocytes and microglial cells extended into the plaques.

Cerebellum.—The atrophy was diffuse and affected all tissue elements, being more pronounced in the left hemisphere and the left part of the vermis than in the corresponding parts of the opposite side. There was, however, no distinct line of demarcation corresponding to the median plane. The pathological changes consisted in a marked reduction in the number of Purkinje cells, especially at the bottom of the gyri, where, in places, they were totally absent (Fig. 6). Most of the surviving

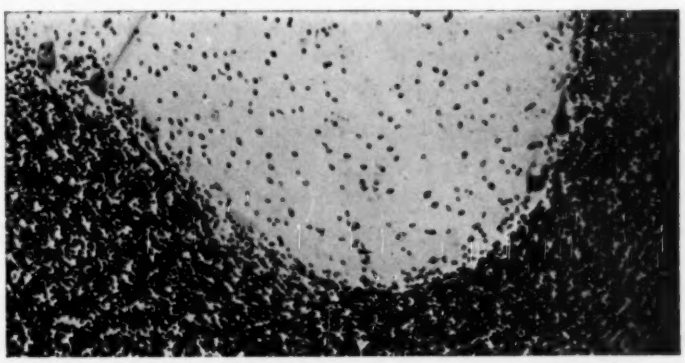

Fig. 6.—Left cerebellar cortex showing loss of Purkinje cells at bottom of cerebellar sulci. Nissl stain.

Purkinje cells showed tigrolysis and shrinking. Silverstain revealed fusiform swelling of the dendrites and axis cylinders of many Purkinje cells. In a number of places where the Nissl stain had shown a lack of Purkinje cells empty baskets were found

Fig. 7.—Left cerebellar cortex showing " empty baskets " of Purkinje cells. v. Braunmühl's silver impregnation.

(Fig. 7). In other places the fibres of the baskets were reduced and the Purkinje cells were present.

There was a small number of argentophile plaques in the cerebellar cortex. The plaques were either of the small solid or the diffuse granular types. The glial elements, especially the astrocytes and the Hortega cells showed diffuse proliferation, more intense in the left hemisphere.

There were no marked changes in the cerebellar nuclei but there was a general cell reduction and marked lipiodosis of the remaining elements. All changes were more pronounced on the left. There was a very obvious reduction of the myelin tissue as a whole and the myelin sheaths showed changes similar to those observed in the cerebral cortex.

Medulla.—No significant changes apart from a lipiodosis of ganglion cells. There was marked diffuse gliosis and cell reduction in the inferior olives, without noticeable asymmetry.

Blood Vessels.—No gross pathological changes. Arteriosclerotic degeneration in some medium arteries ; otherwise the walls of the blood vessels all over the brain were rather thinner than normal. In the cerebral and cerebellar cortex there appeared to be an increase of small vessels and capillaries, an impression obviously due to atrophy of the surrounding tissue.

There was an advanced sclerosis of the choroid plexus in all ventricles, well in accordance with the general atrophy.

Summary of the pathological findings

A remarkably small brain of 870 grm. showing diffuse cerebral and cerebellar atrophy of an unusual degree. The atrophy was of a distinctly asymmetrical character affecting the right cerebral and left cerebellar hemispheres more than the contralateral numbers. The histological examination revealed degenerative changes, i.e. diffuse and local reduction in cortical and subcortical ganglion cells, an enormous number of argentophile plaques with the proliferation of glial elements which usually accompanies the appearance of argentophile

plaques. An uncommonly high proportion of ganglion cells of the cerebral cortex showed Alzheimer's neurofibrillary change which was not found in the cerebellum. There were very marked changes of the axis cylinders in the cerebral cortex with numerous excrescences at right angles to the course of the fibres.

There were degenerative changes in the cerebellum affecting all elements but most distinctly the Purkinje cells. The number of them was considerably reduced especially at the bottom of the gyri where many " empty baskets " were demonstrated. The axis cylinders as well as the dendrites of these cells showed fusiform swelling. All changes were more marked in the left cerebellar hemisphere.

The pathological diagnosis is that of an atypical case of Alzheimer's disease, the most impressive unusual feature being an asymmetrical atrophy of crossed cerebro-cerebellar distribution.

Discussion

The clinical symptoms of the case were rather typical. The post mortem findings confirmed the assumption that the frontal lobes were strongly affected. It is difficult to decide whether the mental deterioration commencing after the birth of a first child born at the late age of 41 is a coincidence without significance, or whether child birth in this case was one of the promoting factors of the disease. However, in view of the lack of present knowledge about such factors this coincidence deserves to be noted. McMenemey (1940) recently discussed this problem.

The kind of the tissue changes, their diffuse but locally more pronounced character and the appearance of the symptoms in middle age are typical for Alzheimer's disease. The degree of the pathological changes in our case was unusual. It is, therefore, of interest that in this case a feature which has attracted less attention, obviously because it is usually little pronounced, was extremely marked, i.e. the particular condition of the axis cylinders in the cortex. It is true that buds and knots on the axis cylinders in Alzheimer's disease were variously described, but they have been usually found on axis cylinders inside and in the immediate neighbourhood of plaques. In our case, most of the axis cylinders of the cortex, both near and at some distance from plaques, showed this abnormality which was absent in areas where there were few or no plaques. The morphological character and the distribution of these changes argue against them being artefacts, i.e. possibly precipitates through the silver impregnation. They probably are the result of a pathological process affecting the argentophile nervous elements and, at the same time, responsible for the formation of the plaque. It is rather tempting to assume that the buds and knots on the axis cylinders consist of the same material as the plaques themselves. Without entering into a discussion on the ætiology of the argentophile plaques—which has been reviewed by Critchley (1929), Wilson (1940) and many others, most recently by McMenemey (1940)—we wish to point out that our case seems to support theories which postulate some process, affecting primarily

the argentophile nervous tissue, as responsible for or essential to plaque formation. Bouman (1934) spoke of a hyperdifferentiation of the argentophile nervous elements. The glial proliferation seemed in our case as in others to be of a secondary character. Our findings do not suggest an intimate relationship between plaques and vascular distribution.

The outstanding feature in the gross pathological findings was the association of an asymmetrical cerebral atrophy with a similarly asymmetrical contralateral cerebellar wasting. The selective nature of the atrophy appeared to be the result of the different degree of the degenerative processes. In our knowledge a predominating crossed cerebro-cerebellar hemiatrophy in Alzheimer's disease has not been described hitherto. Considering the well-known anatomical and functional relationship between each cerebral and the contralateral cerebellar hemisphere the distribution of the atrophy in our case must be regarded as an expression of this correlation.

We do not suppose, however, that the left cerebellar hemiatrophy was entirely secondary to the right cerebral. There certainly was evidence of cerebellar degeneration not dependent on but accompanying the cerebral atrophy. Although in most diffuse degenerative processes affecting the cerebral cortex a certain amount of co-ordinated degeneration takes place in other parts of the brain, e.g. the basal ganglia and the cerebellum, the great difference in the amount of the atrophy of the left and right cerebellar hemispheres in our case must be regarded as resulting from its functional and anatomical relationship to the right cerebral hemisphere. It seems likely that, where a diffuse and circumscribed cerebral atrophy develops slowly a certain amount of cerebellar atrophy usually ensues especially when the cerebral atrophy affects parts of the cortex intimately connected with the cerebellum. But if the cerebral atrophy is less pronounced than in our case and not markedly asymmetrical the cerebellar atrophy is symmetrical and so little obvious that it may escape notice.

As to the aetiology of the asymmetrical character of the atrophy neither the clinical history of our case nor the pathological investigations provides a clue. The following hypotheses may be considered:—

(*a*) The higher degree of degeneration of the right cerebral hemisphere may be the result of a longer duration of the process in that half of the brain. This process may have begun in the right hemisphere some time before it started in the left. An atrophy of the right cerebral hemisphere in a right-handed person may develop slowly for years without causing gross symptoms. Alzheimer's disease commencing in the early fourth or third decade of life or even earlier (Malamud and Loewenberg, 1929) would not be unprecedented. In our case the enormous number of plaques and of cells showing advanced neurofibrillary changes suggest that the process was of extremely long duration, possibly longer than the eighteen years covered by the clinical history. The assumption that the process began early in the right cerebral hemisphere would make the ensuing contralateral cerebellar affection understandable, a result which is more likely to follow in young individuals.

Although an atrophy of asymmetrical distribution is quite uncommon in Alzheimer's disease, it has been encountered in Pick's degeneration. Its

occurrence in our case raises the question whether Alzheimer's and Pick's disease are fundamentally as distinct from each other as they are generally assumed to be.

(*b*) A certain amount of crossed cerebro-cerebellar hemiatrophy may have been in existence before the process of Alzheimer's disease set in. In some cases of infantile hemiplegia resulting in cerebral hemiatrophy slowly ensuing contralateral cerebellar hemiatrophy has been found. (Wertham (1934), Wilson (1940).) That such a process took place in early childhood or even before birth without causing noticeable symptoms or scars in the brain tissue, cannot be entirely excluded.

(*c*) Some process acting more recently than the onset of the dementia and unconnected with Alzheimer's disease may have been responsible for the predilection of one hemisphere. Grünthal (quoted by Wertham) has reported a case in which a trauma had affected one hemisphere long before death and where post-mortem investigation revealed a greater amount of argentophile plaques in the affected side than in the other.

It is not impossible that some cerebral complication of the pregnancy and childbirth preceding the manifestation of the clinical symptoms in our case had made one cerebral hemisphere a punctum minoris resistentiae and that the rapid and extreme atrophy of this hemisphere may have been followed or been accompanied by wasting of the associated contralateral cerebellar half.

Summary

A case of Alzheimer's disease with various uncommon features has been described. The first symptoms appeared after a late pregnancy eighteen years before death. There was an extreme degree of atrophy, the weight of the brain being 870 grm. The atrophy was diffuse but asymmetrical, affecting the right cerebral and the left cerebellar hemispheres much more than the corresponding contralateral halves. There was an extreme degree of the pathological changes characteristic of Alzheimer's disease, the argentophile nervous tissue being most severely affected. The great majority of the pyramidal cells of the cerebral cortex showed Alzheimer's neurofibrillary change. The axis cylinders in the cortex had numerous excrescences. The predominance of the affection of the argentophile nervous elements in an unusually advanced case of Alzheimer's disease seems to argue in favour of the theory that the development of the argentophile plaque is due to a process affecting primarily the argentophile tissue.

The crossed cerebro-cerebellar hemiatrophy is to be regarded as partly the expression of the anatomical and functional correlation of those parts of the brain. The ætiology of the asymmetrical character of the affection has been discussed.

We wish to express our gratitude to Dr. J. J. B. Martin, Medical Superintendent of the Bristol City and County Mental Hospital, for permission to publish this paper.

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THE ELECTRO-ENCEPHALOGRAM IN ACUTE HEAD INJURIES *

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Introduction

IN a series of papers published ten years ago Berger reported abnormally slow waves in the electro-encephalogram (e.e.g.) of patients with diverse intracranial abnormalities. This work has already been adequately reviewed (Jasper, 1937, Walter, 1938). Since then these abnormally slow changes in electrical potential have been investigated in relation to tumours, abscesses, and other local lesions (Walter, 1936, 1937), high intracranial pressure (Williams, 1939), intoxications (Gibbs, Gibbs and Lennox, 1937) and degenerative diseases (Williams and Gibbs, 1939). Similar slow waves are associated with such general cerebral disturbances as the epilepsies (Gibbs, 1939), the psychoses (Hoagland, Cameron and Rubin, 1937) and abnormal personalities (Lindsley and Cutts, 1940).

No such specific characteristics as have been described in relation to the epilepsies have been found in the abnormal slow waves resulting from demonstrable cerebral damage. It appears, on the contrary, that most conditions detrimental to cerebral activity are associated with suppression of the normal frequencies and a concomitant increase in the slower components of the frequency spectrum of the e.e.g. (Gibbs, Williams and Gibbs, 1940). These abnormal conditions range from the genetic abnormality of unstable personalities and epileptics to cerebral destruction by tumours or infection, and include transitory changes in blood constituents or cerebral blood flow. The abnormally slow waves are therefore non-specific, but there appears to be a direct relationship between their number, size, and period and the severity of cerebral change.

During an earlier investigation of intracranial abnormalities (Williams, 1939) an attempt was made to correlate electro-encephalographic changes with different pathological states, and during the course of this work it was found that cerebral trauma produced abnormally slow waves, similar to those already mentioned. These changes seemed to be more variable in extent and degree, and to have a less constant relationship to the apparent severity of the lesion, than did similar changes in other abnormal states. Head injuries are notorious

* This work was communicated to the Neurological Section of the Royal Society of Medicine on June 19th, 1941.

for their variable clinical course, their widespread symptoms, and diverse pathological changes, and consequently for the difficulties in prognosis to which they give rise. When a large number of cases of head injury were made available as a result of the war they were therefore used to study the relationship between the injury, its clinical results, and the consequent changes in cerebral electrical activity. With this in view e.e.gs. were recorded in each case admitted to a special head injury centre. The records were repeated during the period of recovery, the results co-ordinated with other clinical investigations, and experiments undertaken to try to modify the abnormalities found. During the period of this investigation, Jasper, Kershman and Elvidge (1940) described a similar study of sixty-four patients with head injuries resulting from civil accidents, the results of which are in many respects similar to those reached in the present investigation. The present investigation differs somewhat in material and approach, and it is based on observations upon six hundred consecutive cases. The present communication deals with the changes found in those patients in this series who were examined in the acute post-traumatic stage.

Material and Methods

Material.—Seventy-four of the six hundred consecutive cases of head injury were examined by electro-encephalography within 20 days of the injury. Records were taken as soon after the injury as possible, the shortest interval being 50 minutes. The first record was taken within 24 hours in ten, and within the next 48 hours in six, cases. Twenty-four others were examined during the first week, eighteen in the second and sixteen in the third. Fifty-eight of the seventy-four were examined within 14 days of the accident. When the first record was abnormal the e.e.g. was repeated at intervals until improvement ceased. With individual modifications a record was taken daily for the first three days after a head injury, then three were obtained on alternate days, followed by two a week for another month, and thereafter one weekly or fortnightly. In thirty-one of the patients who were repeatedly investigated in this manner, 179 records were taken. Forty-two patients of the seventy-four were found to have a normal e.e.g. initially, and subsequent clinical recovery in them was uneventful. These subjects with a normal record were usually examined only once, so that they account for most of the forty-three patients with single records. The patients were unselected, and all were members of the Army and R.A.F. whose ages ranged from 18 to 60, forty-one being between 18 and 24, and twenty-six between 25 and 30 years of age. The injuries varied from simple concussion to severe cerebral laceration with dural penetration and cerebral herniation. Detailed clinical notes were available in all cases, and in addition a clinical examination, including memory and intelligence tests, was carried out at the time of the e.e.g. investigation.

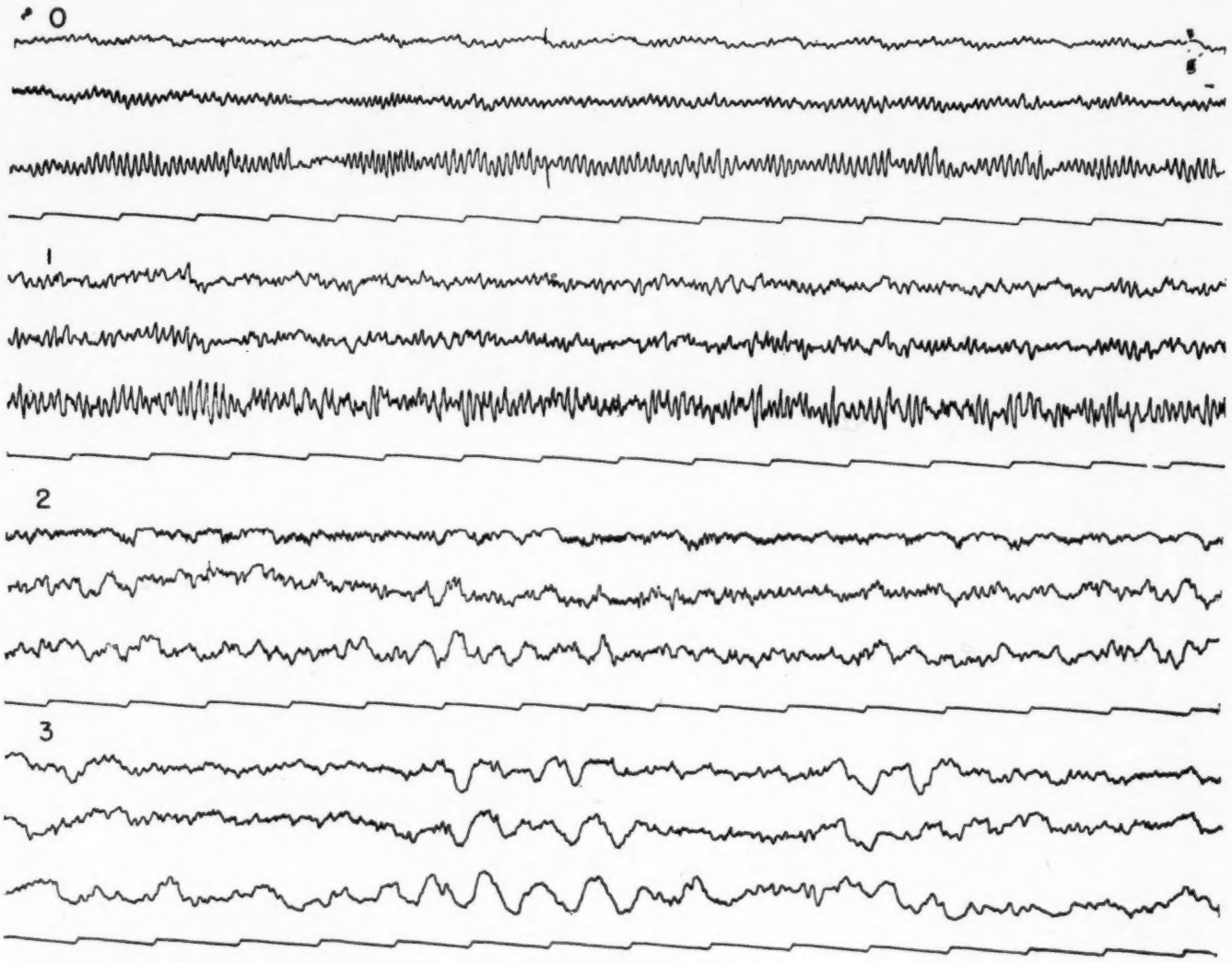
Method of recording.—The patients lay quietly at rest with the eyes closed, on an earthed metal frame trolley in a shielded room. Records were made at various times of the day and night, but when conscious the patients were

invariably awake. Electrodes were placed on the scalp and were connected to three independent condenser coupled amplifiers activating three ink-writing oscillographs (Grass) which recorded simultaneously. Time was marked in seconds.

Electrodes.—Three types of electrode were used. (1) For routine purposes saline pad electrodes were held in position by a rubber and whalebone cap similar to that described by Walter (1937). (2) When an open wound was present the cap could not always be used, so that small solder electrodes were sealed to the scalp with collodium simplex, contact being made with Cambridge electrode jelly. (3) In unconscious patients, or where much local destruction was present, stainless steel needle electrodes attached to fine shellac coated wire were introduced into the scalp or into the lacerated tissue. Whenever needle electrodes were used surface electrodes were placed on the scalp as close to them as possible, and it was apparent that within the limits of the recording system the type of electrode did not modify the e.e.g.

Electrode placing.—In routine investigations sixteen standard positions on the scalp were used, four on each hemisphere in a line from before backwards 3 cm. from the midline, and a similar line of four over the temporal lobe, 3 cm. above the pinna. These electrodes were connected to the recording system, as in Fig. 1. This bipolar method of placing is convenient in routine examination of cases, but many workers (Jasper, Kershman and Elvidge, 1940) use a monopolar system, with an indifferent electrode on the ear. Although the theoretical differences between the two systems are considerable, in practice the records obtained are usually similar. Paired electrodes record the difference in potential between two areas of the cortex, while, theoretically, a single electrode records the absolute potential changes over one area, so that the signal resulting from any one wave arising under one electrode is different in the two systems. In practice the general character of a series of records is not greatly changed, for the wave frequency, voltage relative to other areas, and the proportion of the different wave forms encountered are unchanged. The difference between these systems has already been referred to (Williams, 1938), and their application has been discussed by Kornmüller and Jansen (1939). Bipolar and monopolar leads were sometimes used simultaneously in order to determine from which of a pair of electrodes an abnormal wave form was being propagated, and because the comparison of the data from the two technical methods has an application to the differential diagnosis of intracranial lesions (Jasper, Kershman and Elvidge, 1940). When local changes were found, or where there was clinical evidence of focal cerebral damage, electrodes were placed in and around the abnormal area, their relative positions being changed to suit the immediate need. Thus, in a penetrating gunshot wound of the frontal lobe a line of four electrodes was placed from 2 cm. in front of the hair line, at 2 cm. intervals from before backwards over the wound, the third electrode being a needle introduced into the brain protruding from the wound (Fig. 7).

Clinical examination.—At the time of the e.e.g. the site and extent of superficial cranial damage was noted and abnormal physical signs observed. The



Grade 3. The persistent 1 to 2 per second waves seen in sample 4 have now become intermittent.
Grade 2.—No very slow frequencies are present. They are replaced by intermittent 3 to 4 per second waves of low voltage.
Grade 1. The normal 11 per second frequency now dominates the record, but underlying it are the remnants of the abnormal frequencies, represented by low voltage 3 to 7 per second waves scattered at random through the record. This is the period of incomplete recovery described as " disorganization " or " poor regulation of the alpha rhythm " by other workers.
Grade 0. The e.e.g. is now perfectly normal, with a well sustained 11 per second wave upon a steady base line, full recovery having taken place.

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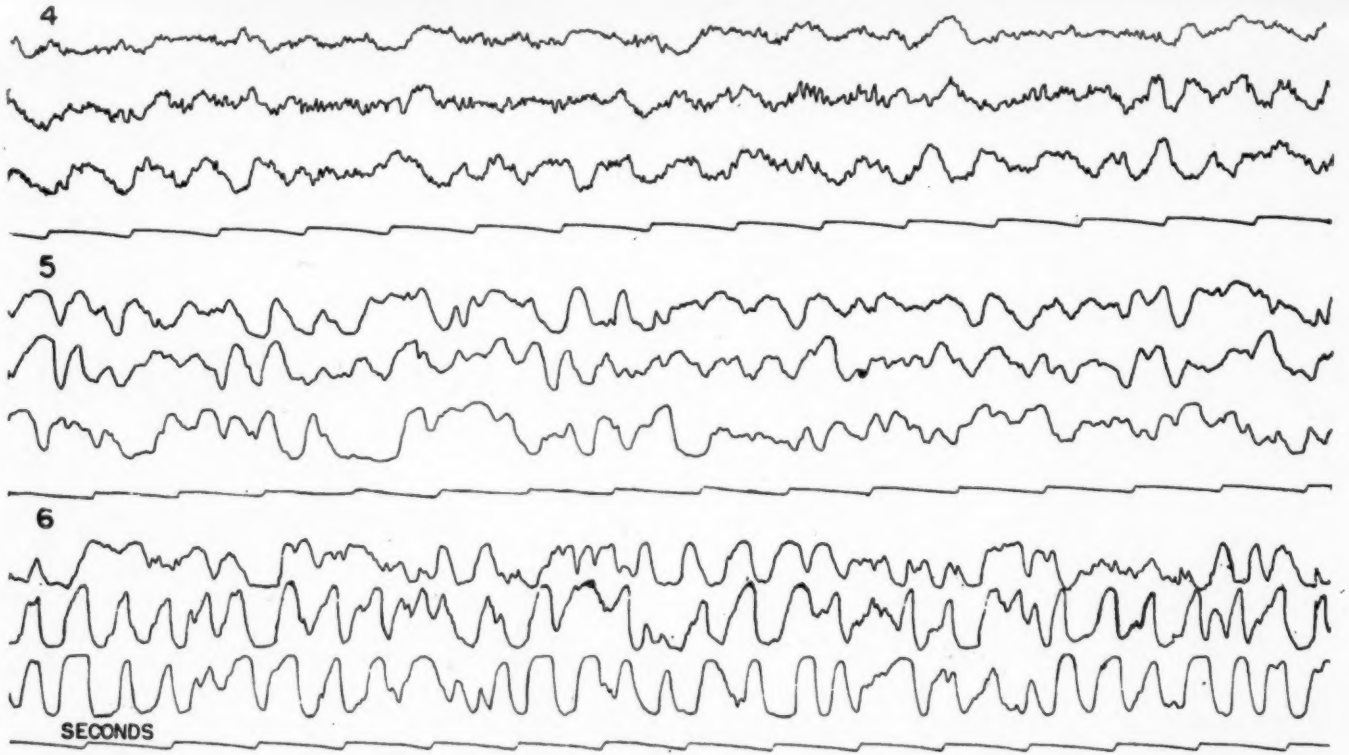

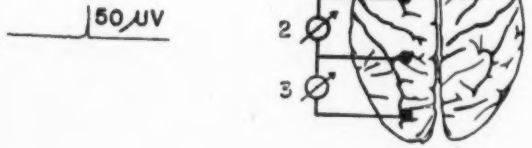

Fig. 1.—Six grades of abnormality of the e.e.g. following severe head injury.

These grades correspond with the degrees of abnormality employed in Fig. 3. Each portion of record contains simultaneous oscillograms from paired frontal, parietal, and parieto-occipital leads. Time is in seconds. The amplification, recording speed, and base-line are constant throughout. The clinical state of the patient is referred to in the text.

Grade 6 taken during coma shows persistent high voltage waves with a frequency of ½ to 2 per second in all leads, with very little faster activity.

Grade 5 contains the same wave forms as seen in sample 6 but their voltage has dropped.

Grade 4 shows persistent rhythmic waves with a frequency of 1 to 2 per second, of lower voltage than in sample 5, superimposed upon which is the returning dominant frequency of 11 per second.

state of consciousness was assessed by a comprehensive method formulated by Group-Captain C. P. Symonds and used as a routine in the hospital. This included observation of the patient's behaviour, mood, insight, and his intellectual functions judged in the light of his past record by his orientation in time and place, awareness of current events, comprehension of complicated requests, memory, powers of calculation, and his judgment. Having obtained these data an attempt was made to correlate the following factors with changes in the e.e.g.: (*a*) The severity of the head injury, judged by the length of post-traumatic amnesia, and the resulting abnormalities, including cerebrospinal fluid changes. (*b*) Bone defects or fissure fractures. (*c*) Dural penetration. (*d*) The time after the injury. (*e*) Previous head injuries. (*f*) The mental state of the patient at examination.

Results

Nature of the abnormal electro-encephalogram. The abnormalities seen were: (1) The appearance of widespread abnormally slow waves of cerebral electrical change, whose frequency varied from the lowest recording limits of the apparatus—less than ½ per second—up to 7 per second (Fig. 1, samples 6 to 2). (2) A suppression of the normal dominant frequencies of 8—12 per second (Fig. 1, samples 6 and 5). (3) Clear-cut outbursts of high voltage sine waves with a frequency of 2—3 per second, lasting for 1—3 seconds. These outbursts were indistinguishable from subclinical epileptic attacks. The characteristics of the abnormal waves did not differ from those described by Jasper, Kershman and Elvidge (Fig. 8).

Slow waves of the first type (Fig. 1, samples 6 to 2) were invariably present when there was impairment of consciousness as a result of the head injury, and their persistence, size, and period were related to the depth of unconsciousness. In cases of closed injury the disturbance was widespread throughout both hemispheres, but occasionally one or more areas of greater abnormality could be distinguished.

Coma was associated with high voltage abnormally slow waves with a period of 1 to 2 seconds and even slower frequencies below the recording limits of the apparatus, with exclusion of any fast activity. This disturbance was always generalized, and is illustrated in samples 6 and 5 in Fig. 1.

Semicoma resulting from head injury was also associated with slow waves whose frequency was higher, voltage lower and persistence less marked than those seen in coma (samples 4 and 3 in Fig. 1).

Confusion, with varying degrees of clouding of consciousness, following a head injury was accompanied by abnormally slow wave forms, which were much more variable in their incidence than those already described. Their voltage and frequency varied from second to second, but short runs of 4 to 6 sine waves, with a frequency of 2 per second, were a characteristic feature (Fig. 1, samples 4 to 2 inclusive). As confusion lightened the runs of slow waves became less evident, the frequency of the abnormal waves increased, while their voltage fell, and the unstable mixture of waves from 3 to 8 per second gradually

disappeared. At the same time the normal frequency of 8—12 per second tended to increase in voltage and to become the dominant feature of the record (samples 2, 1 and 0 in Fig. 1).

The changes in the e.e.g. associated with an uncomplicated head injury are represented diagrammatically in Fig. 2. In this diagram an imaginary curve

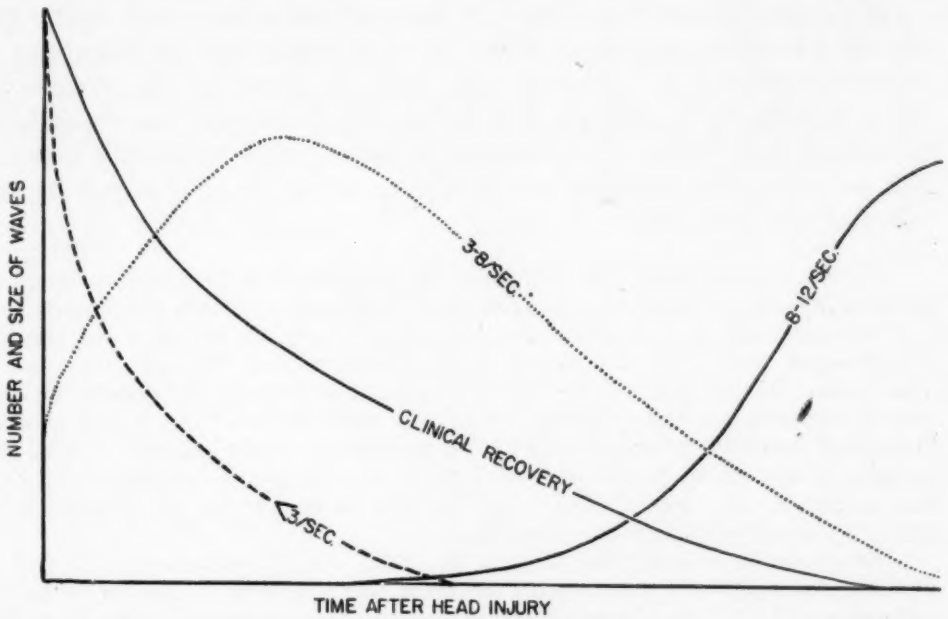

Fig. 2.—A diagrammatic representation of the usual mode of recovery of the e.e.g. in an uncomplicated head injury. A curve of clinical recovery is shown, with the curves of disappearance of the abnormal slow wave forms, and of reappearance of the normal frequencies. The increase shown in the number of medium slow waves is the result of frequency increase of the very slow waves occurring as improvement takes place. The time scale is purposely not specified, for it varies from case to case, the essential character of the curves remaining the same.

of clinical recovery is drawn. In the e.e.g. the very slow waves disappear rapidly, and as they decrease in voltage their frequency increases (Gibbs, Williams and Gibbs, 1940). This speeding up of the slower frequencies implements the number of medium-slow 3—8 per second waves, which subside much more gradually. As the very slow waves are disappearing the normal frequencies gradually begin to return, probably in proportion to their degree of dominance in the pre-traumatic e.e.g. The disappearance of the medium-slow waves lags behind the clinical recovery in many cases, and the superimposition of the dominant 8—12 per second " alpha " rhythm upon them gives rise to the " disorganization of the alpha rhythm " referred to by Jasper, Kershman and Elvidge (1940).

Severity of head injury.—There was a positive relationship between the degree of severity of the head injury and the amount of generalized abnormality in the e.e.g. This relationship has been shown in an analysis of the total series of head injuries (Williams, 1941) and conforms to the observations of Jasper, Kershman and Elvidge (1940), and of Walter (1938), who described a similar

relationship when investigating the effects of tumours upon the e.e.g. Of sixteen patients who had a normal e.e.g. within a few days of the injury, fifteen had a period of post-traumatic amnesia of less than 5 minutes, and the other, who had an amnesia of 10 hours, made a very rapid clinical recovery. Four patients with slight head injuries and a post-traumatic amnesia of less than 5 minutes had a normal e.e.g. within a few hours of the injury, and it remained normal until discharge from hospital. Their clinical recovery was rapid. On the other hand, some subjects with more severe head injuries maintained a grossly abnormal e.e.g. for many weeks, and thereafter the rate of recovery was so variable that it could not be predicted (Fig. 3) except by correlation with the clinical state, which it usually followed closely. The relationship between severity of injury and degree of abnormality of the e.e.g. is shown by the following brief examples:

Mild concussion (Serial Case No. 582). A corporal, aged 20, had a motor-cycle accident 18 hours before an e.e.g. was recorded. Retrograde amnesia was momentary, and post-traumatic amnesia only a few seconds. The patient was alert and normal on admission, and merely complained of local tenderness at the back of the head. The cranium was normal, no abnormal signs referable to the central nervous system were found, and consciousness was not in any way impaired. His behaviour and intellectual functions corresponded with his past record, which had been very good. He had no subsequent symptoms. No fracture was seen on X-ray, and the C.S.F. was normal in all respects. The e.e.g. recorded 18 hours after the accident was perfectly normal and is reproduced in Fig. 4 (a).

Moderately severe injury (Serial Case No. 200). An aircraftman, aged 20, had a motor-cycle accident 20 hours before an e.e.g. was recorded. There was 8 hours retrograde and 12 hours of post-traumatic amnesia. On admission he was alert and co-operative and complained of a slight headache and soreness of the right parietal region. There were no abnormal signs referable to the central nervous system, no bleeding from the ears or nose, but there was bruising of the right parietal area and right ear. The left ankle was fractured. Mentally he was normal to superficial testing. Orientation was correct in place and time, and his general information and judgment were nearly up to his estimated pre-traumatic standard. He only retained 6 digits, but performed the 100-7 test correctly in 50 seconds. An e.e.g. recorded at this time, 20 hours after the accident, is illustrated in Fig. 4 (b). There was generalized abnormality, consisting of large slow waves which occasionally appeared in outbrusts of quite high voltage, the abnormality being greatest in the right parietal region. He continued to have headaches for a week and could not concentrate for long. He improved steadily, and after 2 weeks his intellectual performance and power of retention had improved and he was easily able to retain material read in the newspaper and to discuss it; but he volunteered that he had not his normal grasp of more complicated ideas. He now retained 8 digits and performed the 100—7 test in 30 seconds. There were no abnormal signs at any time. Four weeks after the injury he went to a convalescent home for 10 days and from thence to sick leave and duty. The progressive improvement in the e.e.g. is represented graphically in Fig. 3.

Very severe head injury (Serial Case No. 687). A private, aged 26, had a motor-cycle accident, followed by semicoma. He could be roused by shouted commands and by painful stimulation. There were no superficial injuries. He continued in semi-coma, and 3 days after the accident had 17 left-sided epileptic attacks, followed by a left hemiplegia. When first seen at this time his pulse rate was 52, he was in coma, doubly incontinent, and could not be roused at all. There was œdema of the right parieto-occipital area. He had a flaccid left hemiplegia with absent reflexes and an extensor plantar response, a slight left facial weakness, and the left pupil was

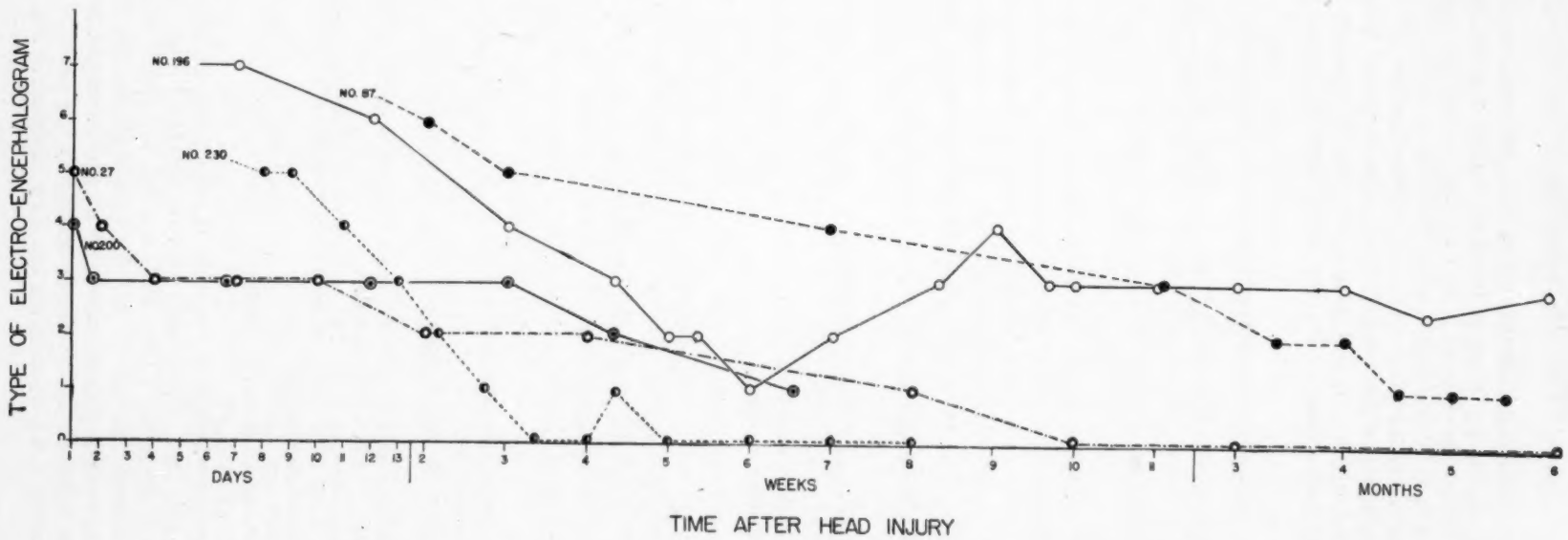

Fig. 3.—Individual recovery curves. The ordinate represents the grades of abnormality in the e.e.g. illustrated in Fig. 1. A different time scale is employed in the abscissa for days, weeks, and months. Protocols of the five cases recorded here are found on pages 127–9.

larger than the right. The C.S.F. pressure was over 300 mm., and the fluid was yellow, but there was no block. X-ray of the skull showed multiple fissure fractures of the right parietal and occipital bones. An e.e.g. on this day showed a generalized disturbance consisting of extremely high voltage rhythmic waves with a period of one second, which persisted without interruption throughout the record. Their voltage was highest in the right frontal and parietal lobes. Improvement followed repeated drainage of C.S.F. and dehydration with mag. sulph., but the pressure remained over 300 mm. for 7 days after his accident. He then passed from semicoma into a gradually

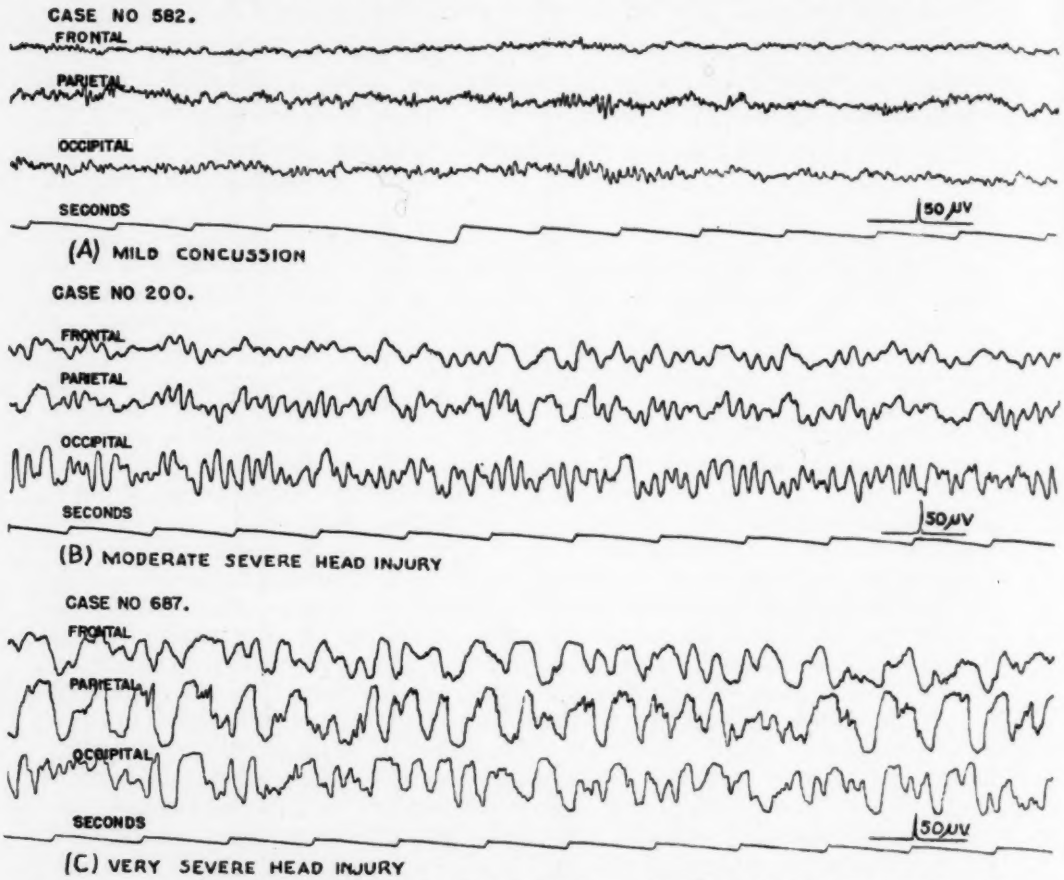

Fig. 4.—In these examples the records are taken from the same electrode placements as in Fig. 1.

(a) The e.e.g. recorded from case No. 582 (page 114), an example of mild concussion, 18 hours after the injury. The record is normal.

(b) The e.e.g. recorded from case No. 200, an instance of moderately severe head injury, 20 hours after the accident. The case history and description of the record are given on page 114 and the case is also represented in Fig. 3.

(c) The e.e.g. recorded from case No. 687, a patient with a very severe head injury, 3 days after the accident. An abstract of the case appears on page 114.

lightening state of confusion, when he was able to answer very simple questions and to perform simple commands. Three weeks after the accident he was alert and could give rational answers to questions. Retrograde amnesia was found to be over a day, and post-traumatic amnesia fourteen days. His hemiplegia steadily improved, and a left homonymous hemianopia gradually disappeared until, 4 weeks after the accident, a small homonymous defect in the left inferior quadrants was the only sequela. He was then mentally normal. A portion of the first e.e.g. is shown in Fig. 4 (c).

Fractures.—Linear fractures of the vault or base of the skull were present in twenty-three of this series of acute head injuries. The initial abnormalities in the e.e.g. and their return to normal in these cases of fracture did not differ from the changes in other cases of equally severe head injuries.

Dural defects and open wounds.—Nine of the cases had penetrating wounds, and in two brain substance was extruding through the wound. This damage caused local changes in the e.e.g., which, as already described, was related to the severity of the cerebral destruction. Although greater abnormality was seen in the area surrounding the opening than was present elsewhere, records taken from the rest of the scalp in these recent severe head injuries always showed abnormality. When needle electrodes were introduced into the wound a focus of discharge could be observed arising from the lacerated brain (Fig. 7). As the e.e.g. and the clinical state improved (*vide infra*) the local abnormality which was initially obscured by the general disturbance became more and more evident until a discrete focus of abnormality could be observed in an otherwise normal series of records.

Time-interval since injury.—The generalized disturbance of cerebral rhythms occurring after head injuries gradually disappeared, and usually the e.e.g. returned to its normal condition. When an attempt was made to plot a curve of average abnormality versus time in the total series it was found that the expected recovery could not be demonstrated. This was because the persistence of the abnormality varied so greatly from case to case that the scattering of individual points was enormous. This can be seen in Fig. 3, which represents the recovery curves in five selected patients. Three of these improved rapidly, and two more slowly, while two of them were discharged before the record had returned to normal, and one even deteriorated after an initial recovery. The improvement in the e.e.g. usually followed the clinical recovery very closely, but in the late stages of recovery there was often a lag when the patient appeared normal to careful clinical testing while some residual abnormality persisted in the e.e.g. This persistent abnormality will be fully discussed in relation to chronic post-traumatic states ; but it was often more spurious than real, for with the clinical methods available the psychiatrist is not able to assess the " functional reserve " of the patient's personality. This was shown by recovery, which appeared perfect in the peace of a convalescent home, giving place to obvious defects in intellectual functions and behaviour on return to the rigours of Service life. Such a response to a change in environment is compatible with the residual abnormality in the e.e.g.

Focal lesions.—The disappearance of generalized cerebral disturbances occasionally unmasked a local area of abnormality. This abnormal area coincided with an open wound when present (Figs. 5 (b), 6, and 7), but focal abnormality was also observed without localizing clinical signs (Fig. 5 (a)). The following three case reports illustrate the occurrence of abnormal discharges from a cortical region also giving abnormal physical signs, and from silent areas, without sequels in one instance and with a general intellectual defect in the other.

Focal abnormality in a silent area without physical signs, but with corresponding personality changes (Serial Case No. 458).—A corporal, aged 50, was run down by a car. He had a laceration of the left eyebrow, with a small fracture below it. Prolonged disorientation made evaluation of retrograde amnesia difficult, but it stretched back for several weeks, and post-traumatic amnesia was for about 4 weeks. He was in semicoma at first and later showed restlessness and negativism. There were no

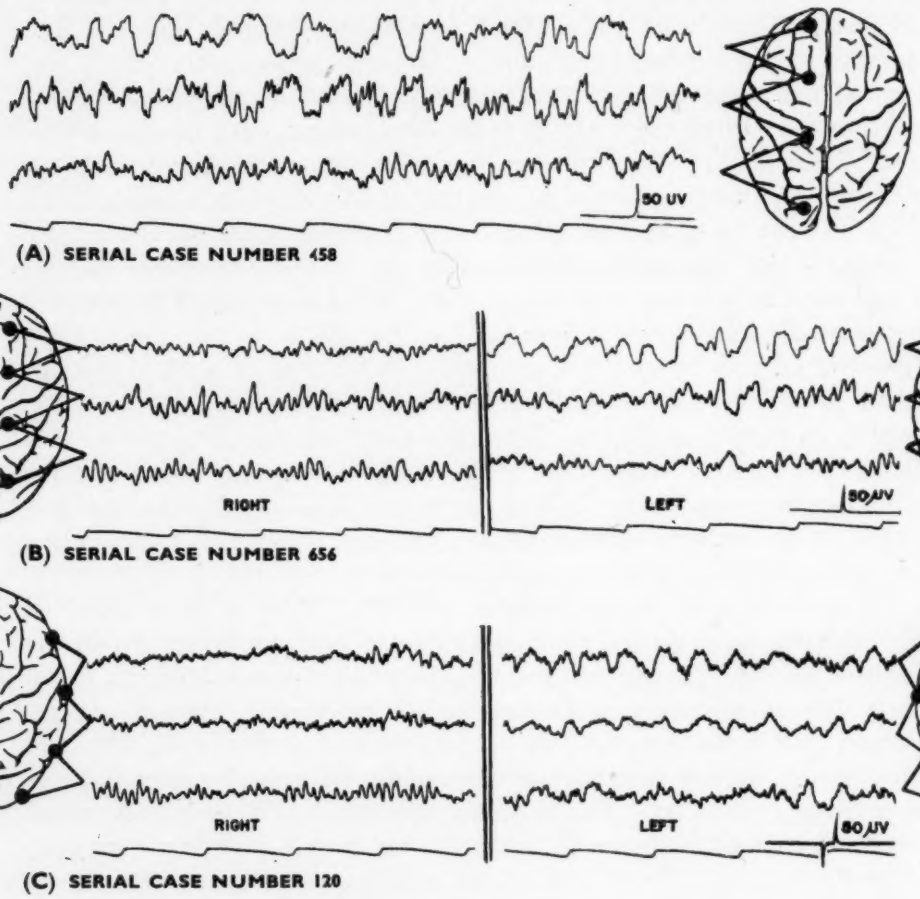

Fig. 5.—Focal Cerebral Damage.

(a) Records from the left hemisphere of case No. 458 (pages 118–9). The high voltage waves with a period of one second are out of phase in the top pair of records, and are not present in the third record, indicating a focus of discharge in the left frontal lobe.

(b) Records from both hemispheres of Case No. 656 (pages 119–20). Those from the right hemisphere are normal, but that from the left frontal pole shows an abnormal rhythm of 3 per second, indicating local cerebral damage.

(c) Records from the temporal regions of both hemispheres of Case No. 120. The records from the left side are normal, but those from the right show a rhythmic 3 per second wave, indicating widespread damage in this region.

abnormal physical signs. Seen 12 days after the accident, he was drowsy, restless, uncoöperative and incontinent. He could be roused by speech, understood commands, and responded to them, but could not retain ideas for longer than a few moments. He was completely disorientated in place and time, had no insight or recall, and confabulated liberally. He would not lie still, and picked at his face and lips continuously. His past record showed him to be a physically healthy but rather

unstable man. The C.S.F. pressure was 190 mm., the fluid was clear, there were 3 cells and 40 mgm. of protein per 100 cu. mm. The e.e.g. showed high voltage abnormally slow waves throughout both hemispheres. Their voltage varied between 100—200 μv. and their frequency from 1 to $\frac{1}{2}$ a second. They were slower in the frontal and temporal regions than elsewhere.

A week later he was more co-operative and less confused, though still restless, incontinent, and completely disorientated. He had a little spontaneous, rational speech. The e.e.g. had improved greatly, except from the frontal lobes, which still showed much abnormality. He continued to improve slowly, and a month after the accident superficially appeared rational.

Five weeks after the accident an encephalogram showed the anterior horns of both ventricles to be dilated. The left was larger than the right, the maximal transverse diameter being 2·5 cm. in the right and 2·8 cm. in the left. He was quiet, docile, and

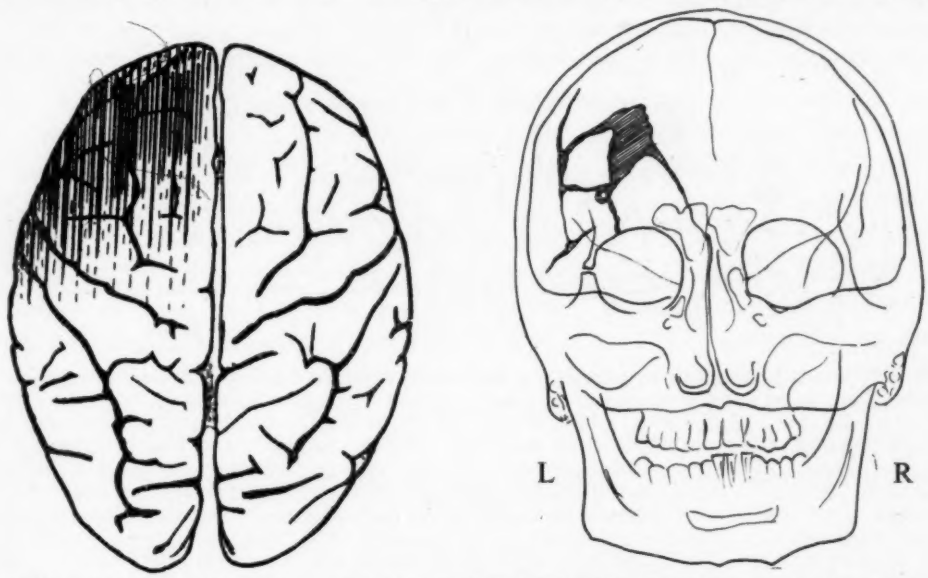

Fig. 6.—A tracing of an X-ray of the skull of Case No. 656, showing an extensive fracture of the left frontal bone, with a defect caused by operative removal of fragments, and a diagram showing the area of brain from which an abnormal e.e.g. was recorded. The situation of the abnormal shaded area corresponds closely with the position of the brain lacerated by bone fragments.

childish. The confusion was much less dense, but he was euphoric, without judgment or insight, and he had no power of recall. He could only retain 4 digits and simple calculation was impossible. Incontinence had ceased. The e.e.g. was almost normal, except from the frontal poles. Here, especially on the left side, there were medium voltage 2 to 3 per second waves (Fig. 5 (a)).

Six weeks after the accident he had improved, but was still confabulating, had no insight, and was euphoric. He could not do the simplest calculation, although recall had improved. Nine weeks after, although the e.e.g. was elsewhere normal, there was still a little residual abnormality from the left frontal lobe. He could then retain 8 digits,took an intelligent interest in international affairs, and had good judgment and insight. He was still euphoric and confabulated about the period of amnesia. Comparison of the changes in the e.e.g. recorded on 8 occasions during the period of improvement in his clinical state showed a close correlation. The clinical picture was preponderantly that of a frontal lobe syndrome, and the e.e.g. showed the abnormality to be greatest in this region.

Focal abnormality confirmed by a bone defect and exploration (Serial Case No. 656).—A private, aged 21, was involved in a motor-cycle collision. Retrograde amnesia

was less than 5 minutes and post-traumatic amnesia about 12 hours. He remembered everything that happened and his only complaint was soreness of the left side of his head. He had no other symptoms of any sort. He had bilateral sub-conjunctival hæmorrhages and bruising but no other abnormal signs, and the central nervous system was normal. X-ray showed a closed depressed fracture of the left frontal bone with a diameter of about 2½ in. A flap was reflected under gas and oxygen, the bone elevated, and several small pieces removed. The dura was torn and there was cerebral laceration. The dura could not be approximated, but the wound was closed in layers. Subsequently there were no symptoms or abnormal signs and the patient made an uneventful recovery. Full psychological testing 12 days after the accident showed no discrepancy between his performance and his record before the injury. An e.e.g. recorded on the same day showed considerable abnormality from an area sharply localized to the left frontal pole. The record from the right hemisphere was within normal limits, but from the left side there was a high voltage 3 per second wave in the anterior pair of electrodes, both in a row of leads near the vertex and in leads along the line of the Sylvian fissure. Fig. 5 (b) illustrates the type of abnormality

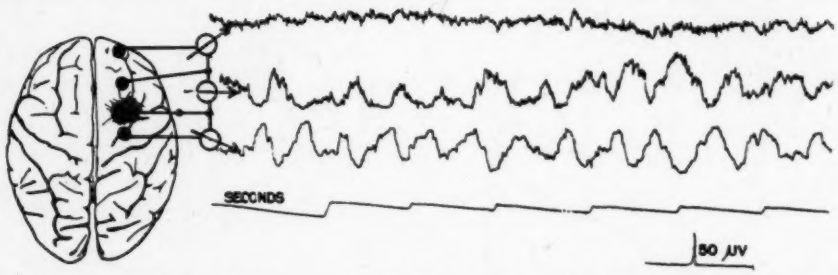

Fig. 7.—An e.e.g. obtained 12 hours after the injury, from the right frontal lobe of a patient with a penetrating gunshot wound of the head. Minute bomb fragments had entered the right frontal lobe in the shaded area, and lacerated cerebral substance was protruding through the small puncture wound. The patient was alert, mentally normal, and had no abnormal physical signs. He had no complaints of any sort. Two surface electrodes were sealed to the scalp anterior to the wound, a stainless steel electrode was placed in the lacerated brain in the wound, and a fourth electrode was sealed to the scalp in line with the others immediately behind the wound. The four electrodes were connected to the three amplifying systems in the manner shown. The record from the first pair consists of a straight base line with fast spikes caused by the activity of the frontalis muscle. The lower two records show an irregular high voltage wave, with a frequency of approximately 2 per second, which shows a persistent phase difference in the two records. This indicates an extremely localized discharge from abnormal brain under the needle electrode, the brain a short distance away being quite normal.

seen, and Fig. 6 shows the correlation between the bone defect and the area of abnormal cortex.

Focal abnormality with corresponding clinical signs (Serial Case No. 120). An aircraftman, aged 20, was struck on the head during a bomb explosion, which he recalls. There was about 24 hours post-traumatic amnesia. He was taken to hospital in a semi-conscious state with a lacerated wound of the left parieto-occipital region. A depressed fracture was present under the laceration, and no exploration was made, the scalp wound above being cleaned and sutured. X-ray showed a depressed left parieto-occipital fracture, with a shower of bone fragments in the underlying cortex. He continued drowsy, had a flaccid right hemiplegia and a right homonymous hemianopia. Three days after the accident he had three grand mal attacks, beginning on the right side. The hemianopia began to clear up in the first week, and then the hemiplegia rapidly improved. He had no further convulsions. The C.S.F. was normal, pressure being 150 mm. He was first seen 3 weeks after the injury. He then had intermittent headaches, and attacks of dizziness on turning quickly. There were no abnormal signs in the central nervous system except for the slightest clumsiness of the right hand. His performance of psychological tests was poor. He retained 7 digits, but judgment,

insight, and intelligence were not of the standard expected from his past record. Encephalography showed a normal ventricular system. He improved steadily, his intellectual level rose, and he ultimately returned to duty. The e.e.g. showed a practically normal record from the right hemisphere, but on the left side there were very well marked abnormal waves with a frequency varying from 1 to 4 per second in the occipital and posterior parietal regions. They were particularly well seen low down behind the ear. A sample of the record and a diagram of the abnormal area are shown in Fig. 5 (c).

One of the outstanding features of e.e.gs. recorded within a few hours of a head injury was the appearance of intermittent outbursts of high voltage sine waves with a frequency of 2 to 3 per second whose character was identical with that of a variant of petit mal epilepsy (Fig. 8). These outbursts usually occurred in all leads simultaneously; but occasionally they appeared in leads from different lobes independently (Fig. 8). They were found when an e.e.g. was obtained within a few hours (up to 48) after a head injury, and in one instance they lasted for 8 days. They were usually associated with an alteration in the state of consciousness of the patient, always subsided completely, and had no apparent relationship to the onset of immediate post-traumatic convulsions. Further, their presence bore no apparent relationship to the subsequent incidence of post-traumatic epilepsy as judged from the follow-up of cases (Williams, 1941). Their presence seemed to be related to the short interval elapsing between the injury and the e.e.g., for they subsided after the acute phase of the post-traumatic state had passed. This type of abnormality is in marked contrast to that seen in other cerebral abnormalities. Neoplasms cause abnormally slow waves, whose period is long in relation to their voltage (Walter, 1937), degenerative diseases are associated with persistent slow sine waves, whose outstanding characteristic is a long period (Williams and Gibbs, 1939), and high intracranial pressure is also associated with persistent slow "rollers" (Williams, 1939*a*). Acute cerebral abscesses are sometimes associated with bursts of relatively high voltage 2 to 3 per second waves, which emanate from the surrounding abnormal cortex, and it is probable that a tumour causing sufficiently severe damage to the surrounding brain will cause similar changes. Even so, it is only in the immediate post-traumatic period and in epilepsy that these outbursts of high voltage slow waves are so clear-cut and characteristic.

Epilepsy.—Six of the seventy-four patients with recent head injuries had grand mal epileptic fits shortly after the head injury. None had had any previous personal or family history of epilepsy, but in one case there was a strong family and personal history of migraine. Two of the six patients had mild, two moderate, and two severe, head injuries. The first fit occurred in these cases 4, 7, and 13 days and 1, 2, and 3 months after the head injury. In two of these subjects records taken between fits were characteristic of epilepsy, as subclinical epileptic outbursts were present. In two the records showed a generalized non-epileptic abnormality, and two were normal. After the changes characteristic of the first few days, no evidence of any epileptic activity was seen in the e.e.g. of any of the cases of recent head injury without fits. The two patients who showed subclinical epileptic outbursts had already had a fit

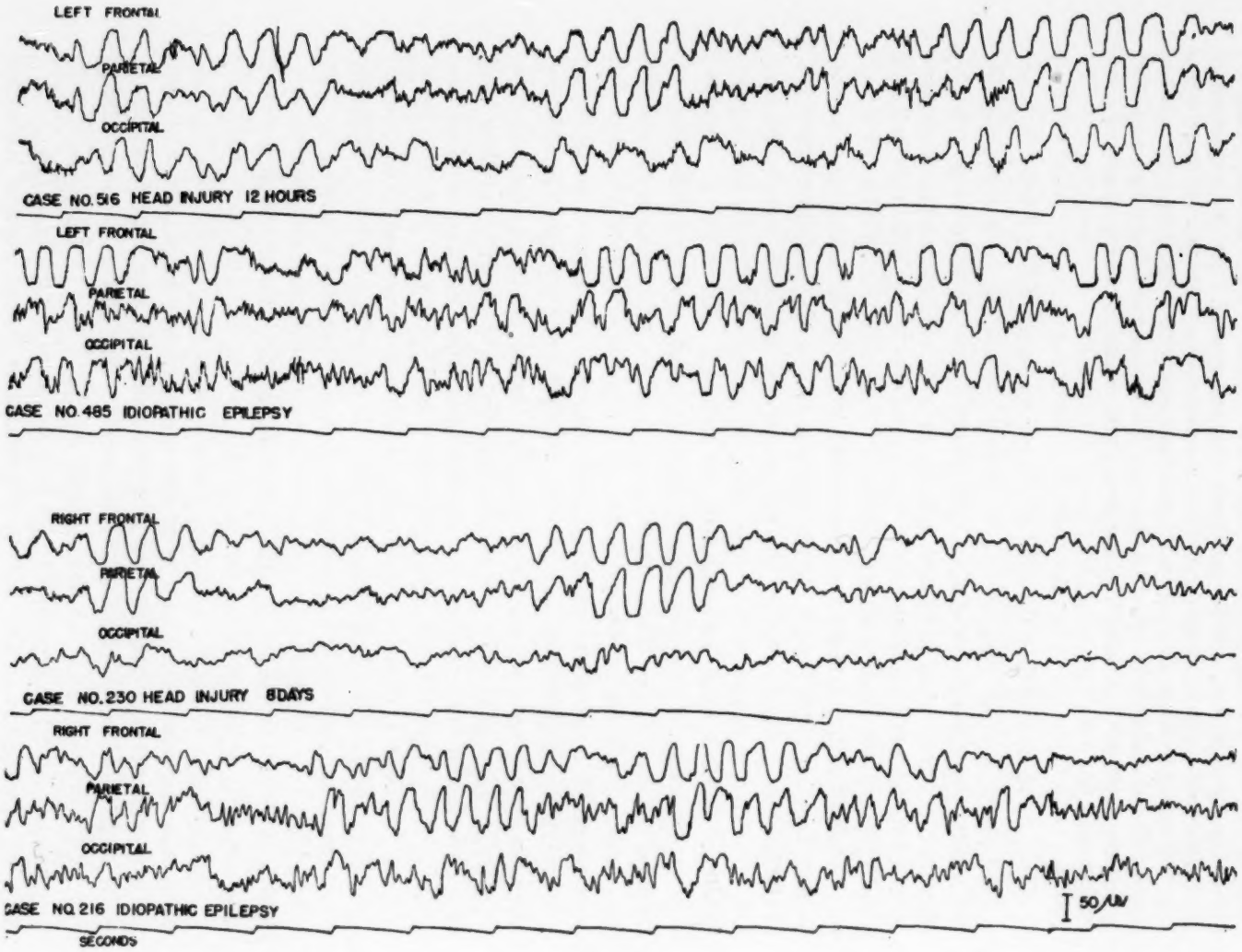
LEFT FRONTAL
PARIETAL
OCCIPITAL
CASE NO. 516 HEAD INJURY 12 HOURS
LEFT FRONTAL
PARIETAL
OCCIPITAL
CASE NO. 485 IDIOPATHIC EPILEPSY
RIGHT FRONTAL
PARIETAL
OCCIPITAL
CASE NO. 230 HEAD INJURY 8DAYS
RIGHT FRONTAL
PARIETAL
OCCIPITAL
CASE NO. 216 IDIOPATHIC EPILEPSY
50 µV
SECONDS

FIG. 8.

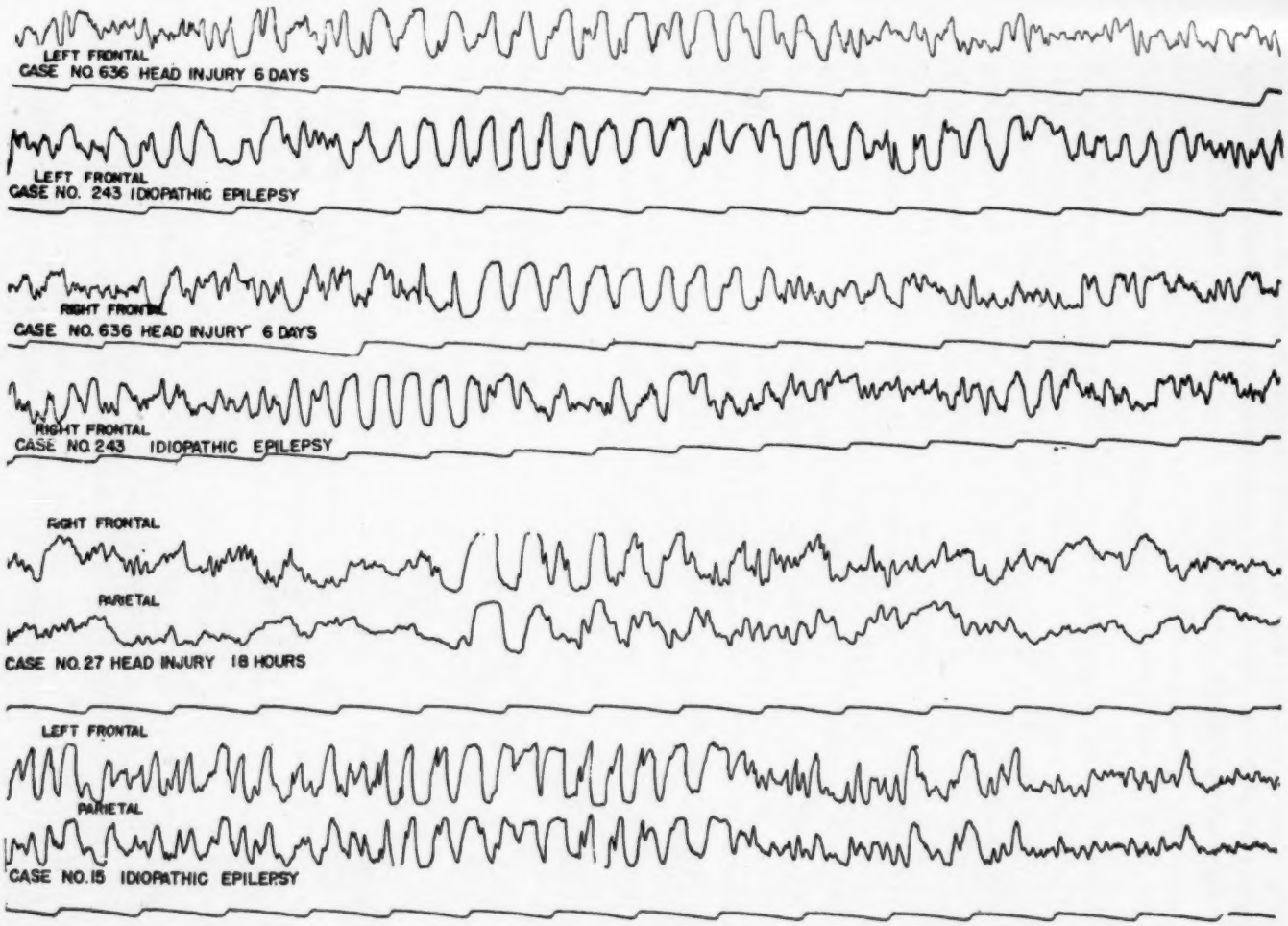

Fig. 8.—A series of e.e.g.'s from four patients within a few hours of a head injury, contrasted with records containing subclinical epileptic attacks from other patients known to have idiopathic epilepsy. None of the patients with head injury showed any evidence of clinical epilepsy at the time of the record or afterwards. The character of the rhythmic outbursts of 2 to 3 per second sine waves and their similarity to subclinical epileptic outbursts are described in the text.

before the e.e.g. was recorded, so that it appears that the onset of post-traumatic epilepsy cannot be predicted by electro-encephalography during the acute phase of head injuries. This subject is discussed in relation to chronic post-traumatic states (Williams, 1941).

Discussion

Although the abnormal slow waves seen in the e.e.g. are non-specific, they almost invariably represent activity arising in abnormal cerebral tissue. This cerebral dysfunction may be associated either with recognizable changes in the neurones themselves, the neuroglia, the intercellular matrix, or be without any demonstrable histological abnormality. Examples of the first type are to be found around a cerebral tumour, a severe cerebral contusion, or in degenerative conditions, such as Schilder's disease. Examples of the second type are to be found among many non-deteriorated epileptics, schizophrenics, and those with abnormal personality traits. Occasionally abnormal records are obtained from apparently normal subjects. These subjects are frequently members of abnormal families and have near relatives with epilepsy, psychoses, or milder disturbances of behaviour (Lennox, Gibbs and Gibbs, 1940); but even if no such history is to be obtained, there is no *prima facie* reason for supposing that because they have so far been normal members of society they will continue so. It is probable that even in this group the abnormal e.e.g. demonstrates a potential abnormality which may unmask itself later in life.

It is important, therefore, to determine if an abnormal e.e.g. discovered after a head injury is the result of the injury or was present before it. In most instances, and certainly in the very acute cases, the degree of abnormality shows that the disturbance is due to the injury, and if the e.e.g. is repeated at intervals and improvement or deterioration in the record occurs, it may safely be concluded that the abnormality is a result of cerebral damage.

The results of this investigation have shown that the abnormalities in the e.e.g. correlate closely with the clinical changes resulting from the head injury, and in this they confirm the observations of Jasper, Kershman and Elvidge (1940). The e.e.g. may therefore be used to follow the progressive cerebral changes resulting from head injuries. The practical value of this is greatest when clinical evidence of damage is slight, for in the immediate post-traumatic phase of a severe head injury routine bedside observation discloses more than the e.e.g. Shortly after a slight head injury, however, the presence or absence of electro-encephalographic abnormality may be a valuable guide to the future management of the case.

As no evidence has been presented, to my knowledge, that active abnormality of cerebral tissue may persist with a normal e.e.g., a normal record after a recent head injury may be taken as indicating the absence of a progressing abnormality of cerebral tissue. Absence of abnormal waves may be associated with complete resolution or absolute destruction of damaged brain substance, for although large cerebral defects following vascular occlusion show abnormally slow waves during the period of degeneration, the e.e.g. ultimately appears

normal, and clean surgical removal of cortex does not leave any aftermath in the e.e.g. It is evident, then, that the e.e.g., when used as an index of active cerebral abnormality after a head injury and correlated with the history and other clinical signs, will help to estimate the extent of the cerebral damage. For instance, a normal e.e.g. obtained shortly after a head injury of whatever superficial character indicates that no gross cerebral damage has occurred, while a normal record in a patient with stupor or confusion attributed to head injury throws doubt on the diagnosis of trauma, for recent damage sufficient to cause obvious impairment of consciousness is invariably associated with an abnormal e.e.g. On the other hand, an abnormal e.e.g. after an apparently trivial head injury may be due either to active cerebral change resulting from the injury or to an abnormal pre-traumatic record, the presence of which, in the light of the observations of Lennox, Gibbs and Gibbs (1940), and Lindsley and Cutt's (1940), upon the high incidence of abnormal e.e.g.'s in potentially abnormal personalities, should lead to a more guarded prognosis than might otherwise have been given. The time taken by the e.e.g. to return to normal after a head injury shows wide variations and unexpected discrepancies, but this again is fully in accordance with clinical experience, and the e.e.g. is merely another objective sign of the rate of cerebral recovery.

In view of the cellular changes which may occur after head injuries it is not surprising that the abnormalities seen in the e.e.g. are widespread. Hassin (1940) points out, from both experimental and pathological studies, that the initial changes following trauma are spread throughout the hemispheres and brain stem; and the early experiments of Scagliosi (1898) and Jacob (1912) clearly demonstrate the diffuse nature of the pathological changes. The abnormally slow waves seen in the e.e.g. almost certainly originate in cortical neurones, either as the result of damage to the cells or indirectly following interruption of projection bundles in the white matter, by section (Bishop, 1936) or by œdema and secondary changes (Williams, 1939). They may even arise as the result of involvement of the brain stem (Walter, Griffiths and Nevin, 1939), which so often occurs in head injuries (Denny-Brown, 1941).

It has been impossible so far to explain the disturbance seen in the e.e.g., in cases of head injury as the result of the direct parenchymatous degeneration (Cajal, 1928), the alteration in fibre pathways (Greenfield, 1938), the generalized œdema (Rand and Courville, 1931), the vascular changes or the petechial hæmorrhages found at necropsy. They are probably all inculpated to varying degrees in different subjects, for Hassin (1940) points out that head injuries cause widespread abnormal physico-chemical changes, whose nature is not yet understood. Trotter (1923) argued that transitory but severe cerebral anæmia might be the prime cause; Cannon (1901) related the disturbance to osmotic changes, while Dixon (1940) has recently suggested that parenchymal hypoglycæmia may result from the commotion of a head injury. Added to these are the more obvious vascular changes, cerebral disorganization by petechial hæmorrhages, and the direct physical violence upon cerebral cells as a result of mechanical agitation (Russell, 1931). The e.e.g. accurately reflects these physico-chemical changes without in any way shedding light on

their character. As consciousness is the result of co-ordination of complex intracerebral mechanisms, the degree of its impairment indicates the general cerebral disturbance following head injury. It is therefore not surprising that changes in the state of consciousness correlate closely with changes in the e.e.g. It must be stressed in this connection that changes in physical signs other than changes in consciousness, X-rays, and C.S.F. findings are too gross indices of damage to be of value in any but the most acute and severe stages of cerebral damage, at which time electro-encephalography is of little practical value.

The appearance of outbursts of abnormal wave forms which are indistinguishable from those found in epilepsy have been reported above, and have been commented upon by Jasper, Kershman and Elvidge (1940). They occur in the acute stage of head injuries and are not always associated with unconsciousness. A review of e.e.gs. taken from a wide range of abnormal states show that these changes are specific to severe cerebral damage and to epilepsy. The observation by Symonds (1935) of the similarity between the phenomena of concussion and the effects of an epileptic explosion is interesting in this respect. Comparing the changes in consciousness following the two conditions he pointed to a similarity between the state of automatism following mild head injuries and petit mal at one end of the scale, and between traumatic stupor and the stupor of epileptic exhaustion at the other, and he said that each grade of cerebral trauma between these extremes could be found to parallel a grade of post-epileptic disturbance. His statement that " whatever is the disturbance of function underlying traumatic unconsciousness, it is probably of the same general nature as that which results from the epileptic explosion " has been given objective support by the present observations.

Only the general disturbance resulting from head injuries has been discussed, but when physical signs are present, an area from which abnormal waves may be obtained will be found to correspond with the predicted site of damage; but local e.e.g. disturbances may be found in the frontal or occipital lobes in the absence of physical signs. A study of these local abnormalities in relation to associated psychological changes may be of considerable value in an investigation of some of the effects of head injuries.

In conclusion it is well to stress again the generalized nature of the electro-encephalographic disturbances resulting from a head injury, which supports the view that cerebral concussion is the result of widespread disorganization of cerebral function and not of specific damage to single structures or isolated tissues.

Summary and Conclusions

Seventy-four patients who had received a head injury were investigated by electro-encephalography within 20 days of the injury. Ten were examined on the first day and fifty-eight within 14 days of the injury. The electro-encephalographic changes were correlated with the clinical state of the patients, judged by physical examination and psychological testing. Open and closed wounds of all degrees of severity were investigated. Widespread abnormally slow waves

of cerebral electrical change, suppression of the normal frequencies, and clear-cut outbursts of high voltage sine waves with a frequency of 2 to 3 per second indistinguishable from subclinical epileptic attacks, occurred in the acute stage.

The degree of abnormality seen in the electro-encephalogram correlated closely with the clinical state of the patient, cases of mild head injury without residual symptoms showing a normal electro-encephalogram within a few hours of the accident and severe injuries causing the abnormalities to persist for many weeks.

The rate of clinical and electro-encephalographic recovery in this series of acute head injuries corresponded closely, but there were wide individual variations in the speed of return to normal. In the later stages of recovery the electro-encephalogram sometimes remained abnormal after the patient had made a complete clinical recovery.

Although the acute changes were invariably generalized, focal areas of maximal abnormality became evident as the general disturbance subsided. When localizing clinical signs were present these corresponded with the area of abnormality seen in the electro-encephalogram. But this local abnormality was also seen in silent areas of the cerebral cortex.

The epileptic type of disturbance occurred in the immediate post-traumatic period and did not have any prognostic significance in regard to post-traumatic epilepsy.

It is concluded that the electro-encephalogram accurately reflects the abnormal cerebral state caused by a recent head injury, that the progressive changes are a direct indication of improvement or deterioration in this state, and that in the milder states of cerebral dysfunction it may demonstrate residual damage after all abnormal clinical signs have subsided. The electro-encephalogram has no value in predicting the onset of traumatic epilepsy in the acute post-traumatic period.

The electro-encephalographic apparatus was lent to the Royal Air Force by the National Hospital, Queen Square, and a grant towards the expense of this work was made by the Medical Research Council.

I am grateful to Lieut.-Colonel G. O. Chambers, M.C. for permission to publish the data included in this paper, to Group-Captain C. P. Symonds and to Colonel Hugh Cairns for extending facilities for the work, and to the Medical Officers whose cases were investigated. I am further grateful to Group-Captain C. P. Symonds and Major D. Denny-Brown for their critical interest in the work and to Miss J. K. Reynell, B.Sc. and Miss C. F. Hatfield for technical assistance.

Clinical Abstracts of the Patients referred to in Fig. 3.

Serial Case No. 27. A pilot, aged 22, was admitted 1 hour after a plane crash, with multiple wounds. Post-traumatic amnesia was 1 hour. He was drowsy but answered questions rationally, and described events up to 5 minutes before the crash. There was an extensive laceration of the forehead, which, with the other wounds, was repaired under general anæsthesia. X-rays did not show any fracture of the skull. Next day he was drowsy and a little irritable. He was correctly orientated and rational but slow in answering. The only abnormality in the central nervous system was slight weakness of the right external rectus 8 hours after the accident.

C.S.F. pressure was 130 mm., and the fluid was heavily bloodstained. The next day he had improved considerably, and 5 days after the accident he retained 8 digits, gave a good account of himself, and showed no intellectual defect. Subsequently he improved rapidly, and was only detained in hospital for management of a Pott's fracture. He had no symptoms or signs referable to the head injury after the first week. The e.e.g. reports are summarized below.

18 *hours*. Records obtained from all positions on both hemispheres were grossly abnormal. There were intermittent runs of high voltage 1 to 2 per second sine waves alternating with periods of lower voltage abnormally slow waves. There was none of the usual dominant frequency. The abnormality was most marked in the frontal lobes (Fig. 8).

2 *days*. The patient was much more alert and rational, and the record showed that the high voltage 1 to 2 per second waves were confined to the left frontal region; elsewhere low voltage abnormally slow waves persisted.

5 *days*. There was a great improvement in the clinical state and the e.e.g. showed corresponding change. The record was much more stable, the abnormally slow waves had lower voltage, were much less obvious, and there were no outbursts of high voltage 2 per second waves from the frontal lobes.

12 *days*. The patient was considered clinically normal. The e.e.g. had improved greatly and the only abnormality was a 5 per second wave of relatively low voltage seen most easily in the frontal lobes, especially the left.

23 *days*. The 5 per second waves were more intermittent and were interspersed with occasional isolated slower waves in an otherwise normal record.

50 *days*. The abnormal 5 per second waves and occasional 3 per second waves persisted upon an otherwise steady base line.

70 *days*. The e.e.g. was perfectly normal and consisted of a low voltage 11 per second frequency upon a straight base line.

This case illustrates the correlation between rapid clinical and electro-encephalographic improvement until a stage was reached when the e.e.g. lagged behind the apparent clinical improvement.

Serial Case No. 87. A private, aged 33, was run down by a car. He was admitted a week later in coma. He was subsequently found to have a retrograde amnesia of 7 days. There were multiple fractures of both frontal bones, which could be moved about *en masse*. He was incontinent, his pupils were dilated and sluggish to light, and there was right facial weakness, a right hemiplegia, and an extensor plantar response. The C.S.F. was clear, the pressure was 100 mm., protein 30 mgm., and cells 7 per cu. mm. The coma slowly lightened, but three weeks after the accident he was still deeply confused, light could not be appreciated in either eye, and the right-sided motor signs did not change. A choroidal tear and primary atrophy were found on the left eye with less marked atrophy in the right. A month after the accident he was still grossly disorientated and confused, his general physical and mental condition had not changed greatly, and he was often incontinent. The pupils were dilated and fixed to light. An encephalogram showed distortion of the right frontal pole, so burr holes were made to exclude a subdural hæmatoma. No subdural or intracerebral hæmorrhage was found, but subsequent X-rays showed a large cyst communicating with the ventricles. Two months after the accident he was still confused, disorientated, had no insight, had little spontaneous activity, and showed marked intellectual impairment. He could talk a little. A month later the mental state had improved considerably, but he was confused for recent events. He was euphoric, had no insight, his conversation was childish and fatuous. The right-sided signs had disappeared. Another month later intellectual improvement was obvious, he took an interest in his appearance, was less facile, and could carry on an intelligent conversation. His powers of recall were still defective. After another month he was invalided from the Army as there appeared to be permanent defects in memory, higher levels of intelligence, and personality.

Serial Case No. 196. A sergeant, aged 24, had a motor-car accident with 6 days retrograde and 6 weeks post-traumatic amnesia. He had multiple cuts on the head and was semi-conscious and restless. He remained semi-conscious, restless, resistive, and noisy. Until admitted to this hospital 3 weeks later he was in traumatic delirium and was restless and resistive. There were no superficial wounds, but he had a slight right facial weakness with more brisk tendon jerks and an extensor plantar response on the left. The C.S.F. pressure was 170 mm., there were 35 mgm. of protein and 3 cells per 100 cu. mm., the fluid being clear. No fracture of the skull could be demonstrated. He remained delirious, confused, and incontinent. A week later he was drowsy, confused, disorientated, and confabulating, although he could retain 8 digits. He remained confused and disorientated for 4 weeks, and during this time he confabulated and was garrulous, euphoric, and uninhibited. The left-sided pyramidal signs had disappeared. Improvement was very slow, but twelve weeks after the injury he was capable of intelligent conversation and was more reserved and stable, his intellectual functions were considered normal, and he could retain 9 digits. He was correctly orientated, his judgment was good, and he had normal insight. His past record and his family history were good, and subsequently he maintained steady progress. At no time were there any blackouts, faints or fits. The e.e.g. showed steady improvement for 6 weeks after the head injury, but then it slowly deteriorated for 3 weeks, and for the last 14 weeks of his stay in hospital it remained at a stationary level of abnormality. The significance of this cannot yet be commented upon, but the patient has so far shown no evidence of any post-traumatic epilepsy.

Serial Case No. 200. An abstract of case number 200 appears on page 116.

Serial Case No. 230. A private, aged 46, while intoxicated was knocked down by a car. Retrograde amnesia was difficult to assess, but was several days, and the post-traumatic amnesia lasted for 12 days. The patient came of a poor, unstable family, and his father and brother were epileptic. He himself was an inferior type of man, with poor intellectual development and little education. When admitted 2 days after the accident he was restless, resistive, and incontinent, he was abusive and profane, but obeyed simple commands correctly, although quite incapable of coherent conversation. There was bruising and a small laceration of the right frontal region, but the central nervous system was normal to full examination. X-ray showed a fissure fracture of the right parietal bone extending through the middle fossa into the base. An e.e.g. showed widespread abnormality, the report read: " There was a great deal of abnormality. The record showed a random mixture of abnormal waves of various frequencies and sizes, but the outstanding characteristic was the presence of outbursts of 2 per second waves of very high voltage, sometimes spreading throughout both hemispheres and sometimes localized to the frontal or occipital poles." (Fig. 8). Four days later unconsciousness was less deep, but he was still confused and disorientated in time and place. There was some dysphasia, but no abnormal signs in the central nervous system. The C.S.F. pressure was 220 mm., the protein content was 40 mgm., and there was 1 lymphocyte per cu. mm. Four days later he was clearer, could remember his station and duties, and the period of amnesia could be assessed. He was still dysphasic. He recovered slowly, but a month after the injury he could only retain 5 digits, his intellectual performance was very poor, and he was truculent, grumbling constantly, and had numerous neurotic traits. His past record suggested that this was his usual pre-traumatic condition, and he was invalided from the Army.

The correlation between improvement in the e.e.g. and in the clinical condition is interesting in this case. His family and past history were so bad and his previous personality and intellectual levels so low that in retrospect it appeared that he had recovered his normal pre-traumatic state much sooner than was realized. Thus the change in the e.e.g. was probably a fair reflection of the actual post-traumatic improvement, which without his past story could not be evaluated clinically.

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THE ELECTRO-ENCEPHALOGRAM IN CHRONIC POST-TRAUMATIC STATES

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THE electro-encephalographic changes following a recent head injury have been described (Williams, 1941). The observations were made upon 74 cases of head injury selected from a total of 600 because the injury had occurred less than 20 days previously. From these observations it was concluded that the electro-encephalogram (e.e.g.) accurately reflects the abnormal cerebral state caused by a recent head injury, that the progressive changes are a direct indication of improvement or deterioration in this state, and that in the milder degrees of cerebral dysfunction it may demonstrate residual damage after all clinical signs have subsided. As symptoms may persist long after full recovery from a head injury might reasonably be expected the whole series of cases was used to observe the subsequent changes in the e.e.g. after the acute effects of the injury had subsided. It is the purpose of this paper to record the results and to relate them to some of the ætiological causes of the chronic post-traumatic syndrome.[1]

Material

Six hundred admissions to a special head injury centre were used. They were unselected except that they were all men serving in the Army or R.A.F. who had symptoms referable to a head injury incurred before or during service. Their ages ranged from 18 to 60 years, but only 35 (5·8 per cent.) were over 35 years old. The investigations were made from 20 days to 12 years after the head injury, the time intervals being grouped below :

TIME AFTER HEAD INJURY	NUMBER OF CASES	TIME AFTER HEAD INJURY	NUMBER OF CASES
Up to 1 month	70	½ to 1 year	41
1 to 2 months	48	1 to 2 years	12
2 to 3 months	43	2 to 5 years	8
3 to 6 months	81	over 5 years	22

[1] It is difficult to give an exact name to the well-known group of symptoms, outstanding among which are headaches, giddiness, defects of memory, lack of concentration, fatigue, irritability, and changes in mood, which arise after head injuries. The old term " post concussional syndrome " has been discarded as inexact and the term " chronic post-traumatic state " is used because its limits are wide. The qualification of " chronic post-traumatic cerebral state " seems undesirable because the syndrome rarely follows injury elsewhere and it is not always due to demonstrable cerebral damage.

Method

The experimental procedure, including the types of electrodes used, their positions on the scalp, and connections to the recording system, as well as the form of clinical examination employed, has already been described (Williams, 1941). Electro-encephalographic recording was made with three independent condenser coupled amplifiers activating three ink writing oscillographs (Grass) which recorded simultaneously, time being marked in seconds (Fig. 1).

Clinical data, which were subsequently correlated with the electro-encephalographic findings, were obtained upon the first 325 patients, and were tabulated under the following headings: age; time after the injury; duration of retrograde and post traumatic amnesia; presence of a cranial fracture, and of an open wound with dural penetration; residual symptoms; fits or faints; personality change; results of psychological examination; abnormal physical and psychological signs; previous head injuries; abnormal family or personal history, with special reference to epilepsy, migraine, constitutional instability, psychopathy or psychosis; the result of treatment, with return to duty or discharge from the service; assessment of the probable causation of the symptoms under three headings—predominantly organic, constitutional, or exogenous psychological factors; and finally the character of the e.e.g.

The assessment of the e.e.g. was based upon records obtained with the patients lying or sitting at rest with the eyes closed, standard amplification, recording speed, and amplifier characteristics being used. The clinical factors to be related to the e.e.g. were so numerous and variable that no attempt was made to quantitate the abnormality in the e.e.g. (Williams, 1941) in each case of this large series. Instead the proportion of abnormals in each group was determined, the norm being based upon the e.e.g. findings in two large control groups of subjects. The criteria of abnormality were similar to those described by Lennox, Gibbs, and Gibbs (1940).

Results

Controls

The first group of normals were 100 flying personnel of the R.A.F. who had undergone rigorous medical selection a few months previously and who had all satisfactorily completed their initial training and were undergoing operational instruction at the time of examination, when their physical health was excellent. Four of these hundred were considered to have abnormal e.e.gs. These four subjects, specimens of whose e.e.gs. are shown in Fig. 1, denied any abnormal personal or family history and may be considered normal by all the usual standards.

The second group consisted of 60 male and female members of the hospital staff. They had passed an army medical examination within a year previously, and their medical records were scrutinized to exclude evidence of abnormality in themselves or their families. In five of these (8 per cent.) the e.e.g. was considered abnormal. The lower percentage of abnormal records in the group of flying personnel than in the nursing staff of the hospital is consistent with their more critical selection. The percentage of abnormality in the hospital staff is of the same order as that obtained by Lennox, Gibbs, and Gibbs (1940) upon a series of non-epileptic controls. An attempt was made to maintain this a constant normal standard throughout the present investigation.

Patients with Head Injuries

Of the 325 patients with head injuries 162 (50 per cent.) had an abnormal

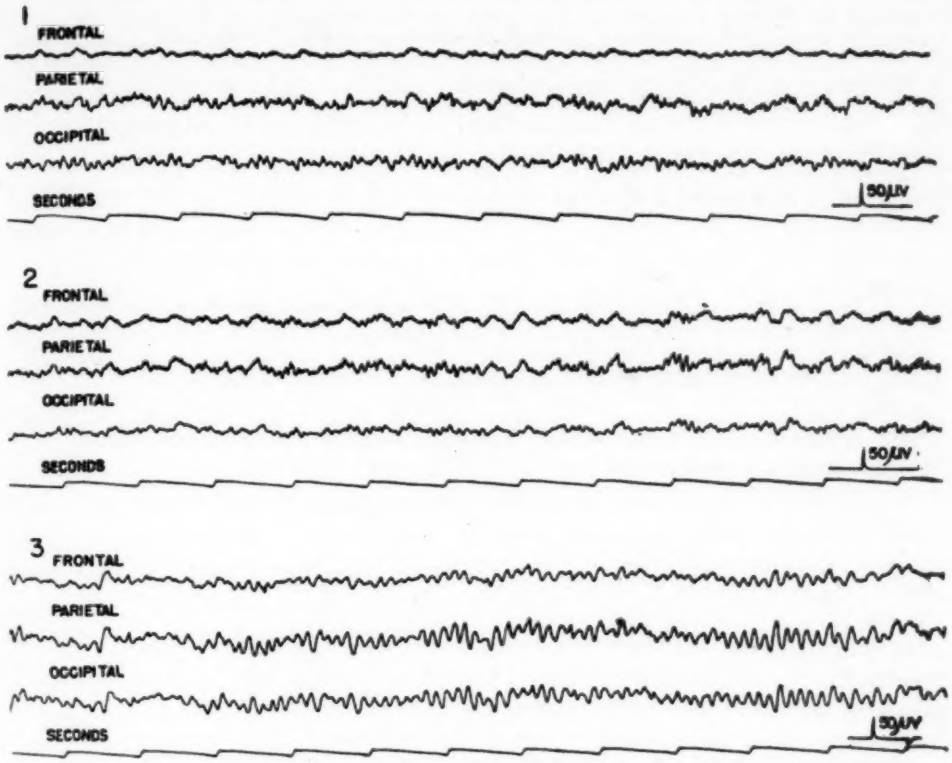

Fig. 1.—Representative records from three control subjects with abnormal electroencephalograms.

e.e.g. As this group of patients was unselected it included patients with injuries, both trivial and severe, as remote as 12 years before the accident. The age group of the patients was the same as that of the normal controls, so that this could not have modified the proportion of abnormal records.

Nature of the Abnormality.—A detailed description of the changes seen in the e.e.g. shortly after a head injury has already been given (Williams, 1941). This description also applies to many of the e.e.gs. seen in the chronic post-traumatic state. The abnormalities seen in this group were, however, less dramatic than those seen immediately after an injury, resembling the later grades of recovery, which have already been amply illustrated. The disturbance was almost invariably generalized, and consisted of low voltage 2 to 7 a second waves underlying the normal dominant frequencies of 8 to 12 and 15 to 25 a second (Fig. 2). After the initial severe abnormalities had subsided during the first few weeks, improvement slowed down and appeared to cease in many of the persistently abnormal cases. The outbursts resembling subclinical epileptic attacks encountered in the immediate post-traumatic period were not seen in non-epileptic patients, nor were very slow waves of high voltage encountered. Thus a uniform type of record, showing mild but persistent generalized abnormality underlying the usual normal frequencies was characteristic of the chronic post-traumatic state (Fig. 2).

Interval after the Injury.—The effect of time upon the incidence of abnormal e.e.gs. after head injuries is shown in Fig. 3. This graph shows that after a

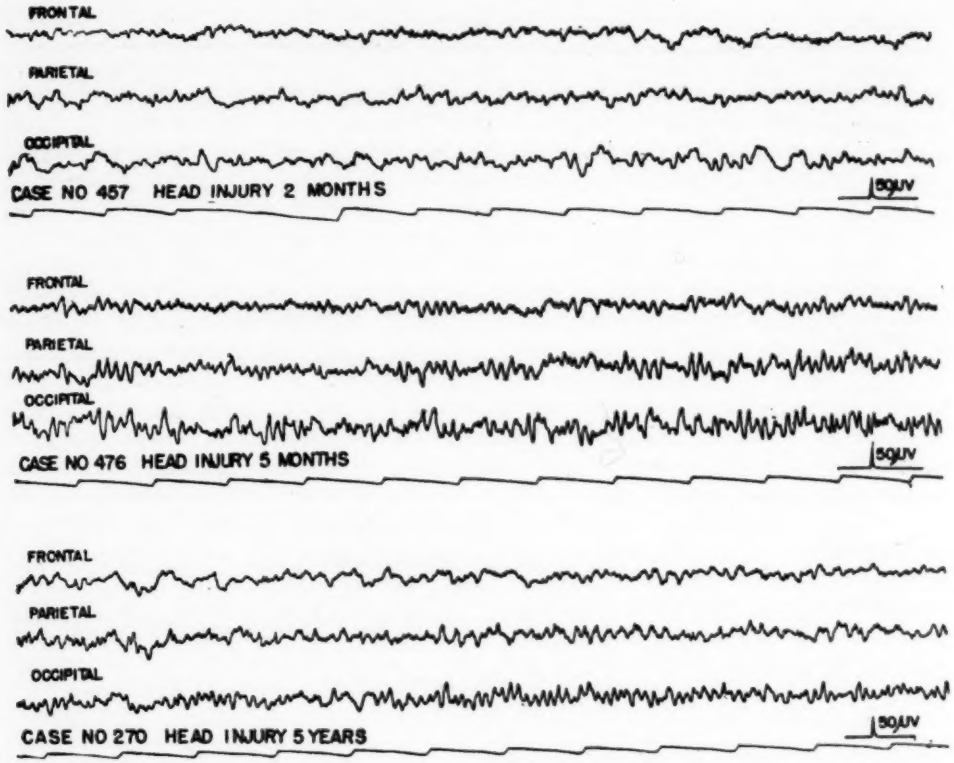

Fig. 2.—Representative records from three patients with abnormal electro-encephalograms 2 months, 5 months, and 5 years respectively after a head injury.

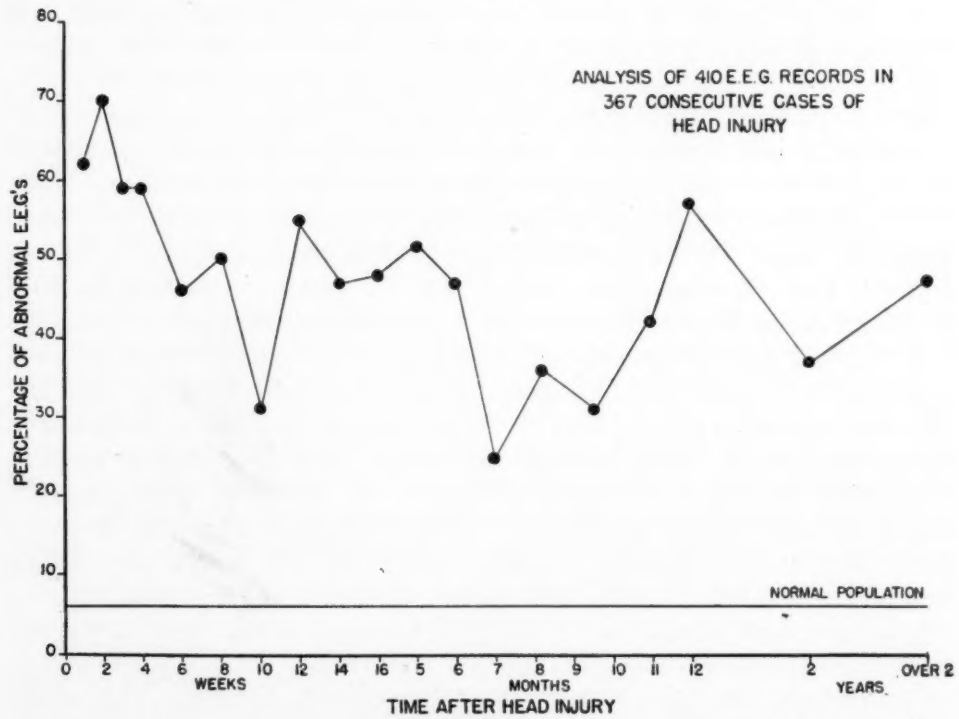

Fig. 3.

60 to 70 per cent. abnormality rate in the first four weeks a decline occurs until ten weeks after the injury. The curve of abnormality then climbs until a further decline takes place at six months. Subsequently there is a steady climb to 12 months, when 55 per cent. of the patients had an abnormal e.e.g. In the main, however, the curve is in the neighbourhood of 50 per cent. and abnormality was still present in 47 per cent. of the cases investigated more than two years after the injury.

Unavoidable selection of cases by their admission to the special centre is responsible for some of the changes seen in the curve. Patients admitted immediately after an injury include many with slight cerebral damage without any prolonged sequelæ, in whom the e.e.g. returns rapidly to normal, concommitantly with the clinical recovery (Williams, 1941). These cases are not comparable with those admitted with symptoms a year after the injury. The climb in the curve between three and six months after the accident is partly due to the admission of many patients who have not recovered after treatment of the acute post-traumatic state in another hospital, many of whom had a severe injury, with a persistently abnormal e.e.g. Again, the level of abnormality throughout the curve is in part determined by the severity and persistence of the symptoms which led medical officers to refer the patients to the centre. Even so it is clear that years after a head injury between 40 and 50 per cent. of patients with symptoms severe enough to warrant admission to a special neurological hospital had an abnormal e.e.g.

Post-traumatic amnesia.—The relationship between the percentage of abnormal e.e.gs. and the length of post-traumatic amnesia is shown in Fig. 4. It is evident that there is a direct correlation between this duration and the percentage of abnormality. As the length of post-traumatic amnesia has a closer relationship to the severity of cerebral damage than any other clinical sign, it can be concluded that there is a positive correlation between the degree of cerebral damage and the persistence of abnormality in the e.e.g. This has special significance when it is considered that 67 per cent. of cases of severe head injury have an abnormal e.e.g., irrespective of the time which has elapsed since the injury. Although this general correlation is close, individual variations occur, and in one instance an abnormal e.e.g. was found without any amnesia.

Retrograde amnesia, whose duration has little relationship to the severity or type of injury was not found to correlate with the changes in the e.e.g.

Open wounds with dural penetration and cerebral laceration were found in 32 instances. The e.e.g. was abnormal in 30 (94 per cent.), the two patients with normal records having been injured 12 and 18 months previously. In these cases a focal abnormality corresponding with the position of cerebral laceration was invariable. although it was usually accompanied by a general disturbance.

Fracture of the skull without opening of the dura was seen in 63 cases, in 30 of whom (47 per cent.) the e.e.g. was abnormal. This figure was not significantly different from that for the whole series, so that the electro-encephalo-

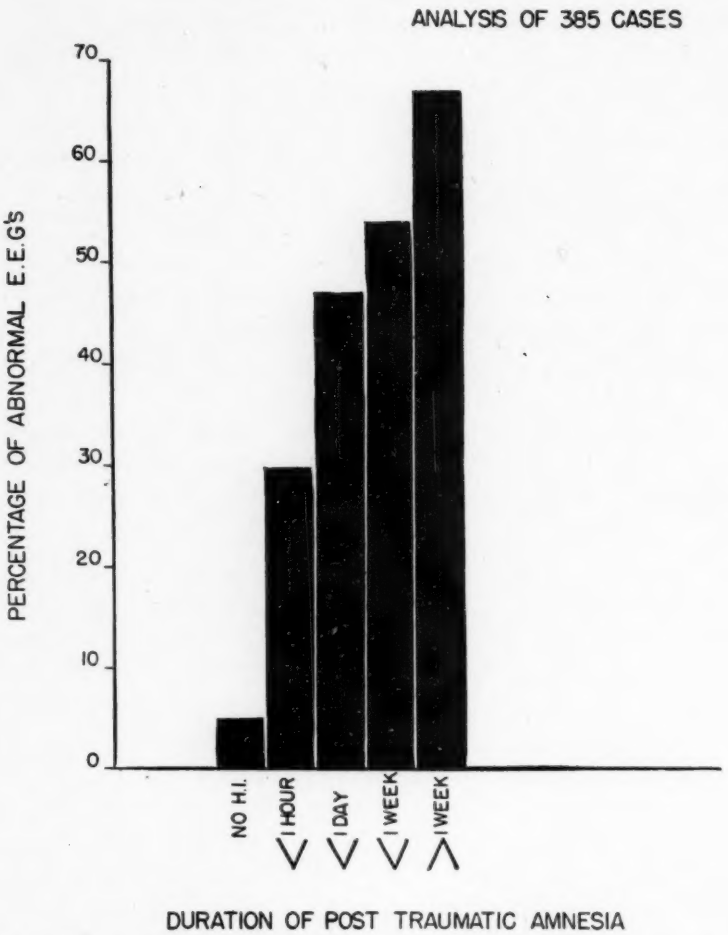

Fig. 4.—The patients with head injuries have been divided into four groups in which the post traumatic amnesia was less than an hour, less than a day, and less or more than a week.

graphic evidence supports clinical experience that a fractured skull does not alone increase the hazards of a head injury.

Persistence of Symptoms.—All excepting 38 of the 325 patients had post-traumatic symptoms when the e.e.g. was recorded. These 38 were either symptom free or had a slight and intermittent headache which soon subsided, and only three (8 per cent.) of these had an abnormal e.e.g. This contrasts with 55·4 per cent. of those with persistent symptoms. It has been shown that in the immediate post-traumatic period the clinical recovery and improvement in the e.e.g. follow each other closely (Williams, 1941). While investigating the later stages of recovery a similar correlation was foumd, and it seemed that interruption of this parallel recovery had bad prognostic significance, for in a symptom-free patient with a normal e.e.g., recovery has already taken place while in a symptom-free patient with an abnormal e.e.g. either cerebral recovery is incomplete or the e.e.g. was already abnormal before the injury. In the first event return of symptoms may occur, and in the second the abnormality in the e.e.g. may be related to an abnormal personality (Lindsley and

Cutts, 1940). An instance of relapse in the first group occurred in Case No. 137.

Case No. 137.—A Gunner, aged 35, was admitted to hospital in coma a few minutes after a motor cycle collision which was subsequently found to have caused between 3 to 4 weeks' post-traumatic amnesia and 2 hours' retrograde amnesia. The next day he was semiconscious and was vomiting repeatedly. There were no signs of local cerebral damage and no wounds. He became restless as unconsciousness became lighter. A week after the accident he was still confused and disorientated, but was improving steadily. Ten days after he felt perfectly well and was anxious to return to duty. His mood was normal, but he was garrulous, euphoric, and uninhibited. On examination he was completely disorientated in time and place, his memory was very bad, and he could only retain 4 or 5 digits. His comprehension, judgment, and calculation were all poor, and he made 6 mistakes in the 100–7 test in 90 seconds. He had no insight and confabulated occasionally. There was slight left lower facial weakness, the tongue was protruded to the left, the tendon jerks were all brisk but symmetrical, all abdominal reflexes were absent, and both plantars were extensor. At this time the C.S.F. was normal, the pressure being 175 mm. of water. X-ray showed a short supratentorial fissure fracture of the occipital bone. At this time the e.e.g. showed widespread and very marked abnormality. There was a high voltage abnormal rhythm, with a frequency of 1 to 2 a second throughout both hemispheres. Underlying this were 3 to 7 a second waves of lower voltage. The disturbance was uniform and symmetrical, and was most evident in the posterior parts of the hemispheres (Fig. 5).

A month after the accident he was still confused and not fully orientated, but he had no abnormal physical signs. Six weeks after he was alert and correctly orientated, and his mental state was improving steadily. The e.e.g. then contained abnormal slow waves of low voltage in both hemispheres, showing a striking improvement upon the initial record. No focal area of abnormality could be found. After two months his mental state was normal except for instability of mood, as he easily became depressed and was unduly worried about the accident. The family and personal histories were good. He had been a steady sober man working as a brick maker before the war. He had a low average intelligence without any abnormal personality traits. His N.C.O. reported that the man was " an excellent and most enthusiastic worker, very popular and well thought of by everyone."

When the patient left the hospital three months after the accident he was considered to have recovered his normal personality and intellectual levels. His memory was thought to be as good as before, although he only retained 6 digits. His general information and calculation were average, his judgment was fair, and his insight and comprehension good. There were no abnormal physical signs. The neurological specialist reported at that time—" I think he has probably made an almost complete recovery, although he may be a little more emotional than previously. His memory is probably up to its former standard. He is fit for duty and I would be very surprised if he cannot carry it out as well as before."

After a month's convalescence he went home to 14 days' sick leave, during which he felt well except for occasional attacks of a postural vertigo which had not been present previously. He returned to his unit anxious to resume his work, but was exhausted by the journey and found that he was " not half the man " he had been. He was given very light work in the billet. The attacks of vertigo began to increase in number and severity, and he had a pressing feeling over the vertex. He rapidly found that he could not cope with his duties, he had no confidence in anything he did, and he felt ashamed of himself. He became anxious, depressed, and emotional. He lost weight and eventually broke down completely and was taken to the medical

officer, who referred him back to hospital a fortnight after resuming work. Psychological examination did not reveal any deterioration since previous testing, and physical examination was negative. He recovered and returned to duty, but after a further two weeks' trial on light work he again broke down, was readmitted, and as his capacity for any but the simplest life seemed limited, he was invalided from the service. Each time the e.e.g. was identical with that recorded before his first discharge from hospital, and it showed a persistence of the abnormal slow waves which had been present previously (Fig. 5).

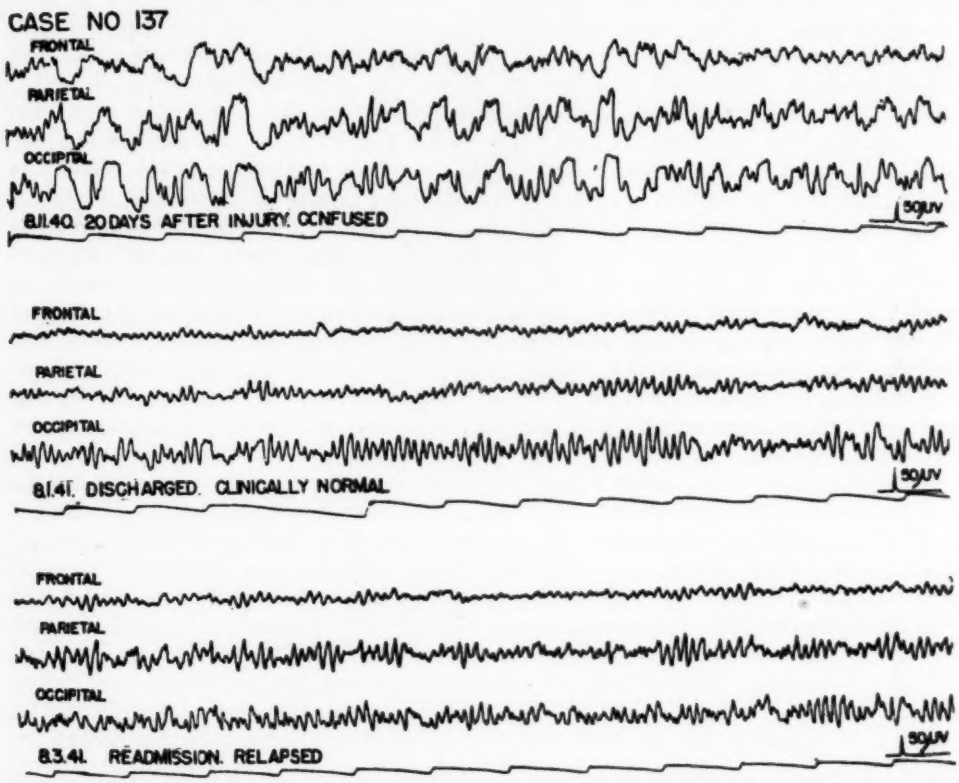

Fig. 5.—Case No. 137. The high-voltage waves with a period of nearly a second recorded 20 days after the injury, have disappeared before discharge from the hospital. The normal dominant frequency has returned, but medium-voltage waves with a frequency of three a second occur frequently, especially in the parieto-occipital region. These abnormal waves were still present on readmission after relapse had occurred.

Comment.—This case report illustrates full clinical recovery after a prolonged period of confusion resulting from a severe head injury, which was followed by a relapse on two occasions when the patient returned to duty. The patient's past record was excellent. The only persistent abnormality was in the e.e.g.

When post-traumatic symptoms had persisted with an abnormal e.e.g., recovery very frequently took place, for cerebral damage giving rise to abnormality in the e.e.g. is usually reversible. As is common experience, the rate of clinical recovery from a head injury is variable, but while the improvement in the e.e.g. is following this recovery resolution appears to be the rule.

When, on the contrary, post-traumatic symptoms persisted with a normal e.e.g. full recovery was not so common. In this series 30 patients with normal

e.e.gs. were invalided because the prognosis seemed so unsatisfactory. The reason for this is apparent if it is accepted that a normal e.e.g. invariably indicates the absence of damaged cerebral substance (Williams, 1941). Post-traumatic symptoms occurring with a normal e.e.g. are either primarily unrelated to cerebral damage or are due to permanent irreversible destruction of cerebral tissue, for absolute destruction of cerebral substance cannot be detected in the e.e.g. unless the involvement is sufficiently extensive to reduce the normal electrical activity. An example of such cerebral destruction with residual symptoms, a normal e.e.g. and incomplete recovery is illustrated by Case No. 331.

Case No. 331.—A Private, aged 25, fell from a lorry on Boxing Day 1940. When admitted to hospital he was in coma and had a spastic right hemiplegia. When he regained consciousness five days later the hemiplegia was unchanged and he was found to have a global aphasia and to be blind in the left eye. X-ray showed a horizontal fracture of the left frontal bone, without any superficial wound. He was admitted to the head injury centre five weeks after the accident, when the following physical signs were found: anosmia, absolute blindness with optic atrophy on the left, ptosis on the left and facial weakness of the right. There was a fairly severe right hemiplegia with spasticity, wasting, and limitation of movement and power, increased tendon jerks, diminished abdominals, ankle clonus, and a flexor plantar response. Sensation was normal. He had a predominantly motor aphasia which was severe enough to prevent psychological testing. The e.e.g. recorded from the right hemisphere at this time was perfectly normal, but there were abnormal slow waves over a very large area of the left hemisphere. Their period was 1 to 2 seconds, and their voltage was moderately high. They were most evident in the parietal and parieto-occipital region (Fig. 6). The C.S.F. was normal in all respects. He improved

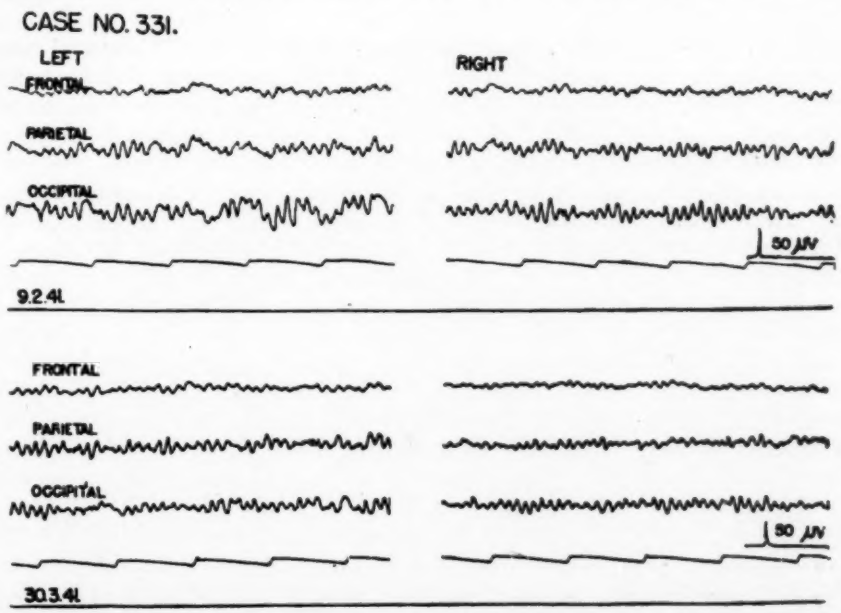

Fig. 6.—Case No. 331. *Electro-encephalogram.* Persistent one-a-second waves were recorded from the left parietal and occipital region 5 weeks after the injury, although records from the right hemisphere were normal. Twelve weeks after the injury, records from identical positions in the left hemisphere were quite normal.

steadily until three months after the accident, when the power and movement on the right side enabled him to walk without support and to feed and dress himself, while the aphasia slowly resolved. At this time intellectual testing showed that he was childish and facile, powers of retention and recall were defective, and his ability to calculate was poor. He was well orientated, had fair insight, but his judgment was poor. An encephalogram showed the anterior horn, body, and temporal horn of the left lateral ventricle to be dilated in all directions, the right side of the ventricular system being normal in size and position (Fig. 7). It was concluded from this and from an increase in the air over the left side of the convexity that there was severe left frontal and temporal cortical atrophy. Specimens of the encephalograms are shown in Fig. 7. Repeated records had been made and had by this time become perfectly normal, all evidence of local disturbance on the left having disappeared (Fig. 6). The patient was observed for over four months after the accident, but at that time there was residual left-sided spastic weakness, dysphasia, and considerable intellectual impairment. Because of the incomplete resolution of symptoms and evidence of gross destruction of cerebral substance the patient was invalided from the Service.

Comment.—This case showed persistence of signs of local cerebral dysfunction with corresponding ventricular dilatation, indicating gross destruction of cerebral issue. The e.e.g. at first showed local abnormality corresponding to the expected site of cerebral damage. This abnormality disappeared, partly perhaps owing to resolution, but largely to absolute destruction of the abnormal tissue. The dissociation between the full recovery in the e.e.g. and persistent symptoms and signs was thought to indicate irreversible destruction of cerebral substance, and an encephalogram subsequently confirmed this opinion.

An example of failure to recover in a patient with persistent post-traumatic symptoms without abnormal physical signs and with a normal e.e.g. is as follows :

Case No. 174.—A Sapper, aged 25, was knocked down by a car and remained semiconscious for two days. Post-traumatic amnesia was later found to be four weeks, and retrograde amnesia a few seconds. He had bilateral extensor plantar responses on admission to hospital. Two weeks after the accident he was restless, grossly confused, and disorientated. There was confabulation and perseveration, he occasionally had organized visual hallucinations, and was incontinent of urine and fæces. There were mild signs of pyramidal damage on the left, with an extensor plantar response, and he had a nominal dysphasia. Lumbar puncture revealed no abnormality except for 80 mgm. of protein. X-ray of the skull was normal. The patient improved very slowly, and a month after the accident was still confused and disorientated. The pyramidal signs had disappeared at this time, but he was still dysphasic. Seven weeks after the accident there was very slight dysphasia. He could retain seven digits but confabulated when attempting to recall simple stories. He had no judgment or insight, could only attempt the most elementary calculations, and made absurd mistakes on being questioned in general knowledge. He slowly improved and ultimately became able to live a normal life, to look after himself, and to carry on spontaneous simple conversations. He remained without insight, and three months after the accident, when he went to a convalescent home, he still had a grossly defective memory, little judgment, only limited knowledge of current affairs, and no insight. He maintained that he was perfectly well. After a month's convalescence he was so anxious to return to duty that he was graded down and returned to simple duties after a month's sick leave. Two weeks after this he was readmitted to hospital, as he was unfit to carry out his light duties in the cookhouse. He complained that the noise of the kitchen had given him intense, continuous headache with tinnitus, and

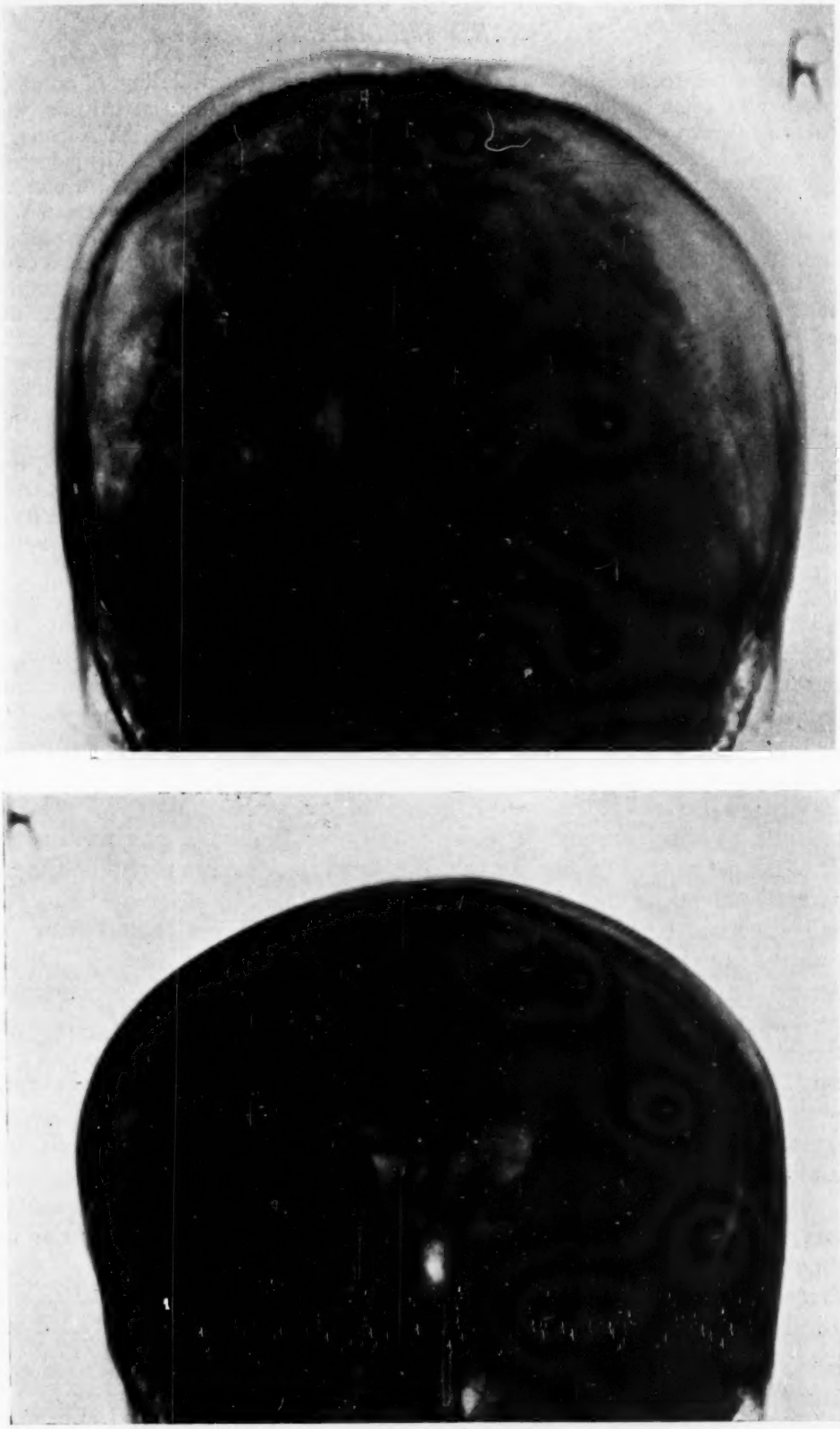

Fig. 7.—*Encephalography.*—Twelve weeks after the injury shows uniform dilatation of the left ventricular system, without displacement of the third ventricle, with a normal lateral ventricle on the right. There is cortical air in the left side, but not on the right. The pictures are taken in the straight antero-posterior and half-axial postero-anterior positions.

he said he had become nervous and anxious. He had persistent feelings of unreality which were often so intense that he thought himself dead. He was irritable, was liable to violent outbursts of temper, and still had occasional visual hallucinations. When readmitted, six months after the injury, he still had no insight. His judgment was defective, and he remained euphoric and wanted to return to duty. He could retain six digits, but could not recount short paragraphs from newspapers, and was unable to obey a series of simple commands. There was obviously mild dementia and considerable personality change. He was invalided from the Service as his condition had been absolutely stationary for three months. Repeated e.e.gs. were normal both before his return to duty and on re-admission to hospital six months after the injury.

Comment.—A man who improved slowly after a severe closed head injury retained considerable intellectual defect with poor memory, little judgment, no insight, and a deterioration in his personality with irritability and outbursts of temper. His condition appeared to be quite stationary six months after the injury. The e.e.g. was originally abnormal, but became normal three months after the injury and did not change subsequently. The disparity between the clinical state and the electroencephalographic findings suggested considerable permanent destruction of cerebral substance. His ultimate degree of disablement supported this view.

Previous Head Injury.—Forty-two patients had had a previous head injury without residual symptoms. Twenty-two of these (52 per cent.) had an abnormal e.e.g., the changes in six being pathognomonic of epilepsy. This series is small, but it suggests that unless epilepsy supervenes, a previous head injury without sequelæ does not appreciably modify the e.e.g. This observation is supported by the presence of normal records in 92 per cent. of the symptom-free patients in this series.

Abnormal Family History.—Evidence of emotional instability, psychoneurosis, psychosis, mental defect or epilepsy was present in near relatives of 73 of the patients. The disorder was considered significant in parents, siblings, parents' siblings, or first cousins. Only 29 (40 per cent.) of these 73 patients had abnormal e.e.gs. and eight of these showed changes characteristic of epilepsy. In view of the low incidence of epileptic changes in the e.e.gs. of the present series of cases (*vide infra*) it is probable that constitutional predisposition was largely responsible for the changes in the eight patients with epileptic records. Excluding the specifically epileptic changes, therefore, only 28 per cent. of this group had abnormal e.e.gs. compared with 50 per cent. of the total. This difference is statistically significant and may have a simple explanation. These 73 patients who came from constitutionally inferior families were probably more liable to have persistent symptoms after a head injury which would not cause sequelæ in more stable patients. Consequently many of them had comparatively trivial injuries, which did not give rise to abnormalities in the e.e.g. Lennox, Gibbs, and Gibbs (1940) have shown that abnormal families have a higher proportion of members with abnormal e.e.gs. than have normal families, so that some of the 29 patients undoubtedly had an abnormal e.e.g. before the head injury.

Abnormal Past History.—Evidence of nervousness, mood swings, psychoneurosis, psychopathy or psychosis was present in 57 patients before the injury ; 29 of these (51 per cent.) had an abnormal e.e.g. after a head injury.

As this proportion is the same as in the total series it might seem that the abnormal past history has no significance, but Lindsley and Cutts (1940) have shown that the e.e.g. is abnormal in a higher proportion of such subjects than in normals so that the pre-traumatic e.e.g. was probably abnormal in some of these subjects, the abnormality resulting from the injury being in fact less frequent than in the total group. In attempting to decide if an abnormal e.e.g. resulted from the injury, records were repeated at long intervals. If the character of the record had not changed it seemed more likely that the abnormality was permanent and so might have been present before the injury.

Disposal of Patients.—Two hundred and seventy-nine of the patients had left hospital when this survey was made. Of 87 who were invalided from the services 67·8 per cent. had an abnormal e.e.g., while only 25·5 per cent. of 192 who returned to duty showed abnormality. It cannot be concluded, however, that a quarter of those returning to duty did so with an abnormal e.e.g., for in most cases the patient went to a convalescent home and then to sick leave after the e.e.g. had been recorded.

Causation.—In attempting to assess the predominant ætiological factor in each example of the chronic post-traumatic state it was realized that in many cases several factors were present simultaneously. It is probable that in some the assessment may have been too strongly biased in one direction, and in others it was even found impossible, but the series was thought to be large enough to minimize individual mistakes. The ætiological factors responsible for the syndrome were divided into three broad groups of organic damage, constitutional predisposition, and exogenous psychological causes, on the basis of the patients' past record, family history, the relationship of the severity of the injury to the resulting symptoms, and the part played by environmental factors in perpetuating the symptoms. An opinion was given upon 316 of the cases ; in 207 the symptoms were thought to be due primarily to organic damage ; 50 patients appeared to be constitutionally predisposed, and in 58 the symptoms were thought to be mainly due to an exogenous neurosis. In the primarily organic group 114 (55 per cent.), in the constitutionally inferior group 19 (37 per cent.), and in the neurotic group 12 (20 per cent.) showed abnormality in the e.e.g.

Epilepsy.—Forty-eight of the 325 patients had one or more epileptic attacks after the head injury ; 32 had grand mal, and in all but two the attacks fell into the group of " immediate post-traumatic epilepsy." Seven patients had a history of fits or faints prior to the injury. Thirty-one of this total (65 per cent.) had an abnormal e.e.g. and the changes seen suggested epileptic outbursts in 20 (42 per cent.) ; eight of the epileptic patients had a family history of epilepsy, and all but one of these had an epileptic type of abnormality in the e.e.g.

Excluding the epileptic type of change seen in the e.e.g. in the immediate post-traumatic period (Williams, 1941) epileptic disturbances occurred without clinical fits in only seven out of 277 patients (2·5 per cent.), thus the percentage of abnormal e.e.gs. (65 per cent.) in the patients with fits, the incidence of characteristically epileptic changes (42 per cent.) in those patients and the

incidence of similar changes in the non-epileptic patients (2·5 per cent.) are all similar to the data relating to idiopathic epilepsy.

Less than a year has elapsed since this series of cases was commenced, so that prognosis cannot be hazarded. It has been shown that the incidence of epileptic changes in the e.e.g. in the epileptic and non-epileptic patients was the same as in similar groups of subjects without head injuries. The character of the changes seen in the e.e.g. record in traumatic epilepsy appear to be the same as in idiopathic epilepsy.

Discussion

This present series does not represent all cases of head injury, for most subjects of concussion or even moderately serious head injury recover without any sequelæ. All except 11 per cent. of this series had sequels of the injury at the time of examination, although the majority (78 per cent.) were investigated after the acute stage had passed. The patients were mostly admitted to a special centre because a severe injury had been sustained, or because recovery was not satisfactory ; but although there was a preponderance of the more severe degrees of cerebral damage the series does represent the group of patients with persistent post-traumatic symptoms. This group presents problems in ætiology, management and prognosis which are not encountered in the milder post-concussional states, so that although a 50 per cent. morbidity in the e.e.g. in no way represents the figure for all head injuries, it is obviously important in relation to these aspects of the chronic post-traumatic syndrome. The importance is enhanced by the persistence of the abnormailty for years after the injury.

There are two possible explanations of this persistent abnormality. It is either a direct result of organic cerebral damage, or it represents an abnormal pre-traumatic e.e.g. in a subject in whom latent abnormality caused symptoms to persist after a head injury. If the second hypothesis were correct the cases would represent the 8 per cent. of clinically normal subjects with abnormal e.e.gs. The results of this investigation show that the proportion of abnormal to normal e.e.gs. was directly related to the severity of injury ; it was higher when the symptoms clinically seemed to be due to cerebral damage than in those patients in whom endogenous or exogenous psychological factors were present ; it was very high when actual cerebral laceration had occurred, while it was low in the absence of symptoms or signs of cerebral damage. Again, the percentage abnormality was not influenced by fracture of the skull, known to be unrelated to the degree of cerebral trauma, nor was it increased by a previous head injury without residua ; lastly, it was no higher in those with a morbid personality or in members of constitutionally inferior families. All these results indicate that actual organic cerebral damage is mainly responsible for the abnormal e.e.g. seen in the chronic post-traumatic states.

It is likely that some of the patients whose symptoms seemed to have been constitutionally predetermined had abnormal e.e.gs. before the injury (Lindsley and Cutts, 1941), but this small number cannot have materially affected the total. It therefore follows from the results obtained in the acute post-traumatic

period (Williams, 1941) that abnormality persisting in the e.e.g. after a head injury is usually due to continued abnormality of cerebral tissue which has resulted directly from the injury. This fact has importance in the management of these cases, for it places a syndrome which superficially has the characters of a " functional " state on to an organic basis. It explains the recurrence of symptoms in some patients after an apparently satisfactory clinical recovery, for the persistent cerebral abnormality reflected in the abnormal e.e.g. cannot be detected by the psychological tests used in routine examination. The residual cerebral damage does not become evident until the patient has to respond to the relatively severe demands of daily employment. Thus undue fatigue, failure of concentration, memory defect and mood changes are related to a loss of " functional reserve " in an organically abnormal brain. This conception is of course an old one, but the changes in the e.e.g. give objective evidence of the organic basis of the chronic post-traumatic syndrome. The evaluation of a normal record in the chronic post-traumatic state is more difficult, for here the symptoms may have arisen as a result of irreversible destruction of cerebral parenchyma or through the perpetuation of symptoms of cerebral damage in an abnormal personality.

It may be impossible on clinical grounds to distinguish the ætiological factor responsible, but whatever the cause of the changes described, the application of the e.e.g. to the clinical data may be summarized as follows :

1. Clinical recovery is followed closely by improvement in the e.e.g. and so long as the improvement in the clinical state and the e.e.g. run a parallel course the outlook is good.

2. Abnormality usually persists in the e.e.g. after clinical recovery appears complete in subjects whose progress is satisfactory.

3. Persistence of abnormality in the e.e.g. usually indicates residual cerebral dysfunction which may not be demonstrable clinically.

4. A normal e.e.g. indicates the absence of abnormal cerebral tissue, so that when associated with full clinical recovery, relapse is unlikely. When symptoms persist in the presence of a normal e.e.g. the ultimate prognosis may be bad, for complete resolution or complete destruction of abnormal cerebral tissue has occurred.

5. No difference is apparent in the e.e.g. of traumatic and idiopathic epileptics.

6. Prediction of traumatic epilepsy does not appear possible by electro-encephalographic examination during the period of recovery from a head injury.

Summary and Conclusions

Electro-encephalograms (e.e.gs.) were recorded on 600 service personnel with head injuries. The interval after the injury varied from a few hours to 12 years, all types of injury being included. The records were divided into normal and abnormal groups on the basis of 160 controls, and the results correlated with detailed clinical data in the first 325 cases.

Fifty per cent. of the patients with head injury had abnormal e.e.gs. This high percentage abnormality persisted for many years after the injury.

There was a positive correlation between it and the severity of the injury, the persistence of symptoms, and the presence of dural penetration.

Simple fracture of the skull, an abnormal family history, an abnormal personal history, and a previous head injury without residua did not increase the percentage abnormality.

Of 87 patients who were invalided from the services, 67 per cent. had an abnormal e.e.g., but of 192 who returned to duty only 25·5 per cent. were abnormal while in hospital.

The cause of the symptoms was assessed. In 207 patients it was thought to be primarily due to organic damage, and in 55 per cent. of these the e.e.g. was abnormal. In 50 who were constitutionally inferior it was abnormal in 37 per cent., and in 58 whose symptoms were due to an exogenous neurosis 20 per cent. were abnormal.

It was concluded that:

(1) After a head injury an abnormal e.e.g. usually indicates organic cerebral abnormality resulting directly from the injury. This abnormality is present in half the cases of the chronic post-traumatic syndrome.

(2) Clinical recovery and improvement in the e.e.g. are usually closely related, and so long as the relationship holds, the prognosis is good.

(3) Abnormality may persist in the e.e.g. in any one patient after clinical recovery appears complete. This indicates residual cerebral dysfunction which is not demonstrable by the usual methods of psychological investigation. It is usually followed by complete recovery, but the abnormality may explain the relapse of some patients after they have returned to their employment.

(4) A normal e.e.g. after a head injury almost invariably indicates absence of abnormal cerebral tissue which may be due either to full resolution or to complete destruction of damaged cerebral substance. When a normal e.e.g. is associated with full clinical recovery prognosis is consequently good, but when accompanied by persistent symptoms the ultimate prognosis may be bad.

(5) There does not appear to be any difference between the e.e.g. of traumatic and idiopathic epileptics, and prediction of traumatic epilepsy does not seem possible by electro-encephalography during the period of recovery from a head injury.

(6) The e.e.g. appears to reflect the degree of cerebral dysfunction resulting from trauma with considerable accuracy, and it has a useful place in the management of cases of head injury when used in conjunction with other methods of clinical examination.

The electro-encephalographic apparatus was lent to the Royal Air Force by the National Hospital, Queen Square, and a grant towards the expense of this work was made by the Medical Research Council.

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Le placement familial des aliénés et des psychopathes. La vie des malades placés. J. Vié. 121.

Le rôle de l'épiphyse dans certaines psychoses. M.-G. Cahana and T. Cahana. 152.

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*Experimental Studies on Headache: Further analysis of the Mechanism of Headache in Migraine, Hypertension, and Fever. A. M. Sutherland and H. G. Wolff. 929.

*Treatment of Unilateral Paralysis Agitans by Section of the Lateral Pyramidal Tract. T. J. Putnam. 950.

*Cortical Softening with Atrophy of Internal Capsule and Dorsal Thalamus: Connections of Ventral Lateral Nucleus of Thalamus. J. W. Papez, H. G. Bull, and W. A. Stotler. 977.

Impulsions: A Specific Disorder of the Behaviour of Children. L. Bender and P. Schilder. 990.

Motor Phenomena During Nitrogen Inhalation. A. Levine and P. Schilder. 1009.

Sensory and Motor Roots of the Glossopharyngeal Nerve and the Vagus-Spinal Accessory Complex. I. M. Tarlov. 1018.

*Effects of Anoxia Induced by Nitrogen Inhalation in Treatment of Psychoses. W. F. Green and J. Adriani. 1022.

*Neuropathologic Study of Six Cases of Psychosis in Which Metrazol Was Used. A. Weil and E. Liebert. 1031.

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Neuropsychiatric Complications following Severe Loss of Blood. N. Reider. 1069.

Diffuse Tuberculosis of the Pituitary Gland Simulating Tumour, with Postoperative Recovery. C. C. Coleman and J. M. Meredith. 1076.

Cyst of the Pulvinar of the Thalamus; Report of a Case with Obstructive Internal Hydrocephalus and Diabetes Mellitus of Intermittent Severity. W. T. Niemer and A. R. Vonderahe. 1086.

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Decerebrate Tonic Extensor Convulsions as a sign of Occlusion of the Basilar Artery: Report of a case with Autopsy. M. Scott and H. C. Lennon. 1102.

Technical and Occasional Notes:

*Cerebral Dysrhythmias in Relatives of Epileptic Persons. L. J. Robinson. 1109.

Mechanism of Headache in Migraine, Hypertension, and Fever.—Experimental studies of headache permit the following inferences: For the histamine headache, it is likely that the cerebral branches of the internal carotid, basilar, and vertebral arteries at the base of the brain are primarily responsible. To the migraine headache, however, the extracranial, and possibly the dural, branches of the external carotid artery are the chief contributors. (R. M. S.)

Treatment of Paralysis Agitans by Section.—Resection of the corresponding transitional cortex at the anterior edge of the Betz cell region gave relief in two cases of unilateral tremor of the hand. Four patients, in whom cervical chordotomy divided completely the lateral pyramidal tract, experienced substantial relief. (R. M. S.)

Cortical Softening with Atrophy of Thalamus.—In a case of complete unilateral atrophy of the thalamus due to vascular occlusion which produced destruction of the cortex and of the internal capsule, the authors have shown that five major afferent pathways end in the ventral posterior and ventral lateral nucleus of the thalamus. These pathways are the spinobulbothalamic tract, conducting cutaneous sensibility; the medial lemniscus, conducting pro-

prioceptive sensibility; the brachium conjunctivum, conducting cerebellar discharge; the fasciculus thalamicus, or field H_1, conducting pallidal discharge; and the intrathalamic fasciculus. Discharges along all five of these pathways find their way to the sensorimotor cortex. (R. M. S.)

Anoxia Induced by Nitrogen Inhalation.—The experience gained with anoxia induced by means of nitrogen inhalation tends to cast doubt on the theory that inference with oxygen metabolism is the modus operandi of insulin and metrazol treatments. (R. M. S.)

Psychosis Treated by Metrazol.—The brains of six patients with dementia præcox, manic-depressive psychosis and involutional melancholia were studied after treatment with injections of metrazol for from 2 to 10 months. The outstanding histopathological features were marked hypertrophy and hyperplasia of astrocytes and, to a lesser degree, of the microglia. (R. M. S.)

Cerebral Dysrhythmias.—Electroencephalograms were obtained from 15 epileptic patients and 36 non-epileptic relatives. Of the former, 80 per cent. had abnormal tracings and in the group of non-epileptic relatives, 36 per cent. showed evidence of cerebral dysrhythmia. (R. M. S.)

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*Inheritance of Cerebral Dysrhythmia and Epilepsy. W. G. Lennox, E. L. Gibbs, and F. A. Gibbs. 1155.
*Neurologic Significance of Platybasia. W. A. Gustafson and E. Oldberg. 1184.
Electroencephalograms of "Constitutionally Inferior" and Behaviour Problem Children: Comparison with those of Normal Children and Adults. D. B. Lindsley and K. K. Cutts. 1199.
*Cerebral Metabolism in Mongolian Idiocy and Phenylpyruvic Oligophrenia. H. E. Himwich and J. F. Fazekas. 1213.
*Pallidohypothalamic Tract, or X Bundle of Meynert, in the Rhesus Monkey. F. Vidal. 1219.
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Vascular Changes in the Thalamic Nuclei Undergoing Retrograde Degeneration. Yü-Ch'üan Tsang. 1237.
A Thirteen Year Follow-up Study of a Series of Cases of Verified Tumours of the Brain. L. M. Davidoff. 1246.

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*An Unclassified Degenerative Disease of the Central Nervous System. W. J. C. Verhaart. 1262.
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Juvenile Familial Amaurotic Idiocy (Vogt-Spielmeyer Disease): Review of Literature and Clinical Report of a Case. S. Levy and O. A. G. Little. 1274.
Cerebral Changes in Rocky Mountain Spotted Fever. G. B. Hassin. 1290.
Aberrant Location of Subdural Hematoma. C. D. Aring and J. P. Evans. 1296.
*Treatment of Trigeminal Neuralgia with Vitamin B_1 (Thiamine Hydrochloride). A. S. Rose and B. M. Jacobson. 1307.

Technical and Occasional Notes:

Element of Optical Illusion in Appearance of Preservation of Axis-Cylinders in Certain Lesions of the Central Nervous System. L. Alexander and T. J. Putnam. 1312.

Cerebral Dysrhythmia and Epilepsy.—Electroencephalographic tracings made on the parents, siblings, and children of 94 patients and on their relatives to the number of 183 suggested that the dysrhythmia of epilepsy is inheritable and that such a dysrhythmia when demonstrable may represent a predisposition to epilepsy or some allied disorder. (R. M. S.)

Platybasia.—Cases of flattened cranial base with deformity of the foramen magnum are discussed in relation to hydrodilatation and other malformations of the nervous system with which they are often associated. (R. M. S.)

Cerebral Metabolism in Mongolian Idiocy.—In mongolian idiocy the brain removes less than the normal amount of oxygen and sugar from each 100 cubic centimeters of blood passing through that organ. The same phenomenon, though to a lesser extent, is observed in phenylpyruvic oligophrenia. Such results might be caused by a decrease in cerebral metabolism or by a faster cerebral blood flow. A review of the literature indicates that the diminished metabolism of the brain is the more probable of the two alternatives. Possible relationship between mental deficiency and diminished cerebral metabolism are discussed. (R. M. S.)

Pallidohypothalamic Tract.—Experimentally produced lesions in the brains of two monkeys indicate that the fibres of the pallidohypothalamic tract have their origin in the globus pallidus. (R. M. S.)

Partial Thenar Atrophy.—Three cases of unilateral partial thenar atrophy are described. They differ from those recently reported in the presence of objective sensory disturbances in all and in the demonstration of sweating disturbances in two. The atrophy in the first case is possibly the result of brachial neuritis. That in the second case is explicable by direct trauma to the nerve. Although there is no history of infection, intoxication, trauma, or pressure in the third case, it is assumed that the atrophy observed in this case, as well as in the others, is due to involvement of selective fibres of the median nerve. Partial thenar atrophy is a benign, nonprogressive condition. It is not related to progressive muscular atrophy, amyotrophic lateral sclerosis, syringomyelia, or tumour of the cord. (R. M. S.)

Alcoholic Cerebellar Degeneration.—The clinical description of five male patients with gradually progressive cerebellar ataxia involving principally the lower extremities

is presented. The onset occurred in middle life, after many years of excessive alcoholic intake. The family histories were without significance. The symptoms were uniform. In addition to the cerebellar ataxia in the lower extremities, nystagmus was present in two patients, speech disturbance in two, and a slight or moderate degree of cerebellar ataxia in the upper extremities in four. There were no evidences of lesions of the pyramidal tract, posterior column, or peripheral nerve in any of the patients. One patient had neurosyphilis, as evidenced by abnormal pupils and a positive Wassermann reaction of the cerebrospinal fluid. The authors believe that the condition of these patients resembles the cerebellar syndrome described as cortical or intracerebellar atrophy. Moreover, they believe that alcohol or an associated nutritional disturbance is a significant factor in the development of this syndrome. (R. M. S.)

Unclassified Degenerative Disease of C.N.S.—The case is described by a Sudanese native aged 45 who had a progressive spastic-cerebellar-ataxic motor deficiency, of about 10 years' duration, which in the later years of his illness changed to muscular weakness, flaccidity, and muscular atrophy, involving the whole body. Post-mortem, there were observed severe degeneration of ganglion cells of the anterior horns of the cord, the motor nuclei of the brain stem, Clarke's column, the pallidosubthalamiconigral system, the basal pontile nuclei, and the dentate nucleus. There was no swelling of ganglion cells, inflammation, primary demyelination. formation of abnormal glia cells or signs of abnormal metabolism in the central nervous system. This case is compared with those of other atypical degenerative diseases that have been described in small numbers in the literature. No etiological agent could be established. (R. M. S.)

Treatment of Trigeminal Neuralgia with Vitamin B.—The exhibition of vitamin B (thiamine hydrochloride) in four cases of trigeminal neuralgia had no beneficial effect. (R. M. S.)

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*Petechial Hemorrhages of the Brain Experimentally Produced in Rats by Concussion. W. F. Schaller, K. Tamaki, and H. W. Newman. 1.

*Arsenic as a Possible Cause of Subacute Encephalomyelitis: Correlation of Chemical, Clinical, and Histologic Observations. A. D. Ecker and J. W. Kernohan. 24.

Distribution of Nerve Terminals (Boutons) in the Human Spinal Cord: Quantitative Studies. J. Minckler. 44.

*Changes in the Brain in Alcoholism. L. D. Stevenson, L. E. McGowan, and A. M. Allen. 56.

*Studies in Diseases of Muscle: ix. Effect of Quinine and Prostigmine Methyl Sulfate on Muscular Rigidity in Paralysis Agitans. A. T. Milhorat. 74.

*Seeping Intracranial Aneurysm Simulating Neoplasm: Syndrome of the Corpus Callosum. W. H. Sweet. 86.

Representation of the Sympathetic and Parasympathetic Nervous Systems in the Forebrain of the Cat. H. B. Carlson, E. Gellhorn, and C. W. Darrow. 105.

*Cerebral Swelling and Edema Associated with Cerebral Tumour: A Histogenetic and Histopathologic Study. I. Scheinker. 117.

*Mescaline Hallucinations in Artists. W. S. Maclay and E. Guttmann. 130.

*Connections of the Medial Geniculate Body in the Cat. H. W. Ades. 138.

Case Reports:

Rhythmic Myoclonus: A Clinical Report of Six Cases. J. P. Dobson and H. A. Riley. 145.

Juvenile Huntington's Chorea. E. I. Falstein and T. T. Stone. 151.

Clinical and Electroencephalographic Changes in a Child During Recovery from Encephalitis. D. B. Lindsley and K. K. Cutts. 156.

Petechial Hæmorrhages of Brain.—By a special device white rats were subjected to a propulsion impact injury to the head, similar to the mechanism of head injuries in man frequently occurring in automobile accidents. The vascular pathological lesions described by the authors are considered to be due to specific concussion effects and differs from contusion, laceration, and tearing, in that they are the result of vascular dilatation, stasis, and anoxæmia. (R. M. S.)

Arsenic as Possible Cause of Encephalomyelitis.—In 12 cases of subacute encephalomyelitis the clinical, pathological, and chemical findings were consistent with the hypothesis that they represented instances of chronic arsenical poisoning. (R. M. S.)

Brain Changes in Alcoholism.—In this study the pathological changes in the nervous system were relatively slight as compared with the severe and fatal illness of the patient and the authors therefore believe that the changes usually responsible for death and for the clinical picture in chronic alcoholism cannot be demonstrated under the microscope by methods now at one's disposal. Most of the changes are probably due to avitaminosis B_1 and B complex rather than to alcohol itself. (R. M. S.)

Diseases of Muscle.—Quinine and prostigmine methyl sulphate were observed to have effects in some patients with paralysis agitans that were similar to those seen in patients with "myotonia." Hence the assumption that the effects of these drugs indicate the nature of the defect in "myotonia" appears unjustified. It is probable that the effect of quinine on muscular function in cases of "myotonia" and paralysis agitans is the result of the antagonistic action of the drug to cholinergic nerve stimulation. (R. M. S.)

Seeping Aneurysm Simulating Neoplasm.—Two cases of seeping aneurysm of a cerebral artery simulating cerebral tumour

are reported. In the first the aneurysm caused a softening confined almost exclusively to the corpus callosum. This lesion produced apractic agraphia and ideomotor apraxia in the left hand, and its extension into the most medial portion of the left hemisphere probably explains the forced grasping that was present in the right hand. Forced grasping may be the first sign of invasion of the opposite hemisphere by a cerebral tumour. (R. M. S.)

Cerebral Swelling and Cerebral Tumour.—In cases of cerebral œdema the author described the typical and well-known picture of the areolar, sievelike appearance of the nerve tissue and distension of the pericellular and perivascular spaces, with resulting liquefaction of the nerve tissue. Particular attention is drawn to the morphological signs of alteration of the circulatory system with increase in the permeability of the vessel walls. In cases of cerebral swelling such morphological changes in the vessels are absent. In this condition there are mild functional vesicular disturbances only (stasis and diapedesis), and usually the capillaries alone are involved. (R. M. S.)

Mescaline Hallucinations.—Hallucinations during mescaline intoxication cannot be explained in either physiological or psychological terms alone. The fact of hallucinating and some formal characters of the hallucinations are so similar to physiological and pathological phenomena that they can be assumed to be physiological in origin, but psychological experiences determine the contents of the hallucination; for example, it can be said that the appearance and repetition of similar shapes are caused physiologically, but the facts that the subject sees women and not men and that the scotomas take on the shape of lotus flowers can be accounted for only in psychological terms. (R. M. S.)

Connections of Medial Geniculate.—Recurrent fibres pass from the medial geniculate body to the inferior colliculus and to the trapezoid body by way of the brachium of the inferior colliculus and the lateral lemniscus, respectively, and thus provide a system of reverse innervation between subcortical acoustic nuclei. Diffuse fibres leave the medial border of the medial geniculate body and pass to other parts of the thalamus and midbrain. No evidence is found to support the contention that the medial geniculate body has any important function as an acoustic reflex centre. The commissure of Gudden does not connect the two medial geniculate bodies. Fibres pass from the medial geniculate body to a cortical area bounded anteriorly by the anterior ectosylvian sulcus, posteriorly by the posterior ectosylvian sulcus, superiorly by the suprasylvian sulcus and inferiorly by the superior extremity of the pseudosylvian sulcus. This area coincides with that responsive to acoustic stimulation. (R. M. S.)

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Vol. 45. No. 2. February 1941.

*Experimental Studies on Headache: A. Contrast of Histamine Headache with the Headache of Migraine and That Associated with Hypertension: B. Contrast of Vascular Mechanisms in Preheadache and in Headache Phenomena of Migraine. G. A. Schumacher and H. G. Wolff. 199.

Observations on Reflex Responses During Prolonged Periods of Human Refrigeration. T. Fay and G. W. Smith. 215.

*Memory Defects During Metrazol Therapy. E. Ziskind. 223.

*Tumours of the Brain in Aged Persons. F. P. Moersch, W. McK. Craig, and J. W. Kernohan. 235.

*Mechanism of Displacement of the Ventricular System in Children with Atrophic Lesions. B. Crothers and G. M. Wyatt. 246.

An American Family with Pelizæurs-Merzbacher Disease. C. D. Camp and K. Lowenberg. 261.

Ingestion of Large Doses of Dilantin Sodium. C. D. Aring and M. Rosenbaum. 265.

*Elicitation of the "Pseudomotor Contracture" in the Tongue by Intramedullary Stimulation. K. B. Corbin, F. Harrison, and C. Wigginton. 271.

*Sugar and Oxygen Metabolism of the Brain During and After Insulin Hypoglycemia. J. Loman. 282.

Effects of Insulin and Metrazol Therapy on Cerebrospinal Fluid Proteins. G. , Kingsley and H. Freed. 289.

Rôle of Trauma in Amyotrophic Lateral Sclerosis. R. W. Waggoner and K. Lowenberg. 296.

*Lymphogranulomatosis (Hodgkin's Disease) of the Nervous System. N. W. Winkelman and M. T. Moore. 304.

An Adaptation of the Sayre Apparatus for Use in Encephalography. E. H. Read. 319.

Case Reports:

Infraction of the Brain with Unilateral Circulatory Changes. C. Pilcher, T. E. Wyatt, and H. M. Carney. 321.

Contrast of Histamine Headache and Migraine.—Histamine headaches were abolished by increasing the intracranial pressure, thereby giving extramural support to the cerebral arteries at the base of the brain. This is further evidence that histamine headaches result mainly from the dilatation and distention of cerebral arteries. The headaches of migraine were not reduced in intensity by increasing intracranial pressure. This is further evidence that such headaches arise from extracerebral arteries. Headaches associated with hypertension were not reduced in intensity by increasing intracranial pressure. This is further evidence that such headaches have a similar origin in extracerebral arteries. Scotomas occurring as preheadache migraine phenomena were demonstrated to result from constriction of cerebral arteries. (R. M. S.)

Memory Defects during Metrazol Therapy.—Four cases of severe and 20 of slight amnesia developed with metrazol treatment. The memory abnormality is related to the number of metrazol convulsions. Recovery is the rule. (R. M. S.)

Tumours of Brain in Aged Persons.—In a series of 100 cases of verified tumours of the brain occurring among patients 60 years of age or more, glioblastoma multiforme, meningioma, and acoustic neuroma, in the order named, made up 82 per cent. of the total. The cerebrum was the most common site of development of tumour, tumours in this region constituting 69 per cent. of the series. (R. M. S.)

Displacement of Ventricular System in Children with Atrophic Lesions.—If both hemispherea are damaged and fail to grow important dislocation rarely takes place, regardless of asymmetry. Since growth is most rapid in the prenatal period and in the early months of independent life, the finding of maximal dislocation when the injury occurs in early life is easily explained. If dislocation is the result of growth, failure of dislocation in the presence of gross asymmetry of the ventricles, in early childhood at least, may be a sign of bilateral failure of cerebral development. No evidence was found to support the hypothesis that traction can pull the midline structures and the contralateral ventricle toward an atrophic lesion. The mechanism of displacement may be the same, whether the pressure comes from unresisted thrust from a growing hemisphere or from an expanding lesion. (R. M. S.)

Elicitation of "Pseudomotor Contracture."—In order to locate the origin of fibres mediating the lingual pseudomotor contracture (Philipeaux-Vulpian-Heidenhain phenomenon) and to determine the cranial nerve over which these fibres leave the brain stem, many points in the pons and mesencephalon have been stimulated in 25 cats with chronic sections of the hypoglossal nerve. Stimulation of the mesencephalic root of the fifth nerve or of its mandibular division never gave rise to pseudomotor contracture, as has been claimed by a few investigators. The pseudomotor contracture was elicited by stimulation of the intramedullary portion of the seventh nerve, the reticular matter close to it, and the chorda tympani fibres peripherally. Stimulation of these structures also elicited vasodilatation. Section of the chorda tympani nerve abolished the response to intramedullary stimulation; section of the proximal portion of the lingual nerve did not influence the response. The criteria for determining a true pseudomotor response are discussed and the possibilities for error pointed out. The function of the fibres mediating this response is discussed. (R. M. S.)

Sugar and Oxygen Metabolism and Insulin Hypoglycæmia.—Studies on subjects with dementia præcox of the passive type gave no evidence that sugar and oxygen metabolism of the brain is stimulated after insulin hypoglycæmia. On the contrary, neural dysfunction, rather than stimulation of cerebral respiration, may be the important factor which is related to the change in mental states of subjects treated by insulin hypoglycæmia or other forms of shock. (R. M. S.)

Insulin and Metrazol Therapy and C.S.F.—Both insulin and metrazol caused appreciable changes in the cerebro-spinal fluid proteins of schizophrenic patients. The immediate effect of metrazol was a lowering of the albumin-globin ratio. Later, after 1 to 8 weeks of treatment and a rest of 3 to 4 days, the albumin and albumin-globulin ratio increased, while the flobulin decreased. Insulin caused a rise in globulin and a depression of the albumin-globulin ratio; when the treatment was discontinued the albumin and albumin-globulin ratio returned to higher values, while globulin decreased. (R. M. S.)

Hodgkin's Disease.—Two cases of Hodgkin's disease with involvement of the nervous system have been reported in detail. In case 1 there was clinical evidence of involvement of the brain, left brachial plexus, and spinal epidural space. Histological examination of the brain showed extensive intracerebral lymphogranulomatous foci and dural plaques. In case 2 clinical and pathological evidence of invasion of the mid-thoracic spinal epidural space by a Hodgkin's process was presented. The histopathological picture in both cases was typically that of Hodgkin's lymphogranuloma. The distribution and character of the cerebral lesions indicated a blood-borne invasion. The authors' cases, as well as those in the literature, point to the necessity for careful neurological study in all cases of Hodgkin's disease, in view of the frequency with which the nervous system is involved. (R. M. S.)

Vol. 45. No. 3. March 1941.

Histologic Evidence of Damage to the Brain in Monkey's Treated with Metrazol and Insulin. K. H. Finley and C. Brenner.

Amphetamine Sulfate-Sodium Amytal Treatment of Schizophrenia. E. Davidoff, E. C. Reifenstein, and G. L. Goodstone.

Sweat Mechanism in Man: Study of Distribution of Sweat Fibers from the Sympathetic Ganglia, Spinal Roots, Spinal Cord and Common Carotid Artery. O. R. Hyndman and J. Wolkin.

Neoplasm of the Posterior Fossa Simulating Cerebral Vascular Disease: Report of Five Cases with Reference to the Rôle of the Medulla in the Production of Arterial Hypertension. B. C. Meyer.

Metamorphosia and Other Psychovisual Disturbances in a Patient with Tumour of the Brain. M. B. Bender and M. G. Kanzer.
Convulsive Seizures in Delirium Tremens. M. Rosenbaum, M. Lewis, P. Piker, and D. Goldman.
Cysticercosis of the Brain: Report of a Case with Operation. B. S. Ray.
Chemistry of Anticonvulsant Drugs. T. J. Putnam and H. H. Merritt.
Differential Diagnosis of Hysterical Tremor. P. Schilder.

Case Reports:

Astrocytosis Arachnoideæ Cerebelli: A Rare Manifestation of von Rechlinghausen's Neurofibromatosis. A. Earl Walker.
Danger of Subarachnoid Injection of Alcohol for Relief of Pain. R. A. Groff and F. H. Lewy.

Vol. 45. No. 4. April 1941.

*Neurologic Syndromes Accompanying Developmental Anomalies of Occipital Bone, Atlas, and Axis. C. F. List. 577.
*Convulsive Phenomena Produced by a New Method of Remote Excitation. F. A. Fender. 617.
*Prolonged Jugular Compression: A Diagnostic Test of Neurologic Value. R. B. Aird. 633.
*Clinical Trial of Beta Erythroidine Hydrochloride in Hypertonic and in Hyperkinetic States. V. W. Eisenstein and M. Tarlau. 649.
*Poliœncephalitis Hæmorrhagica of Wernicke Associated with Febrile Disease: Report of Two Cases, One Occurring During Typhoid Fever and Verified by Autopsy. Y. C. Chang, T. H. Suh, and C. C. Ling. 658.
*Corticomeningeal Scars in Traumatic Epilepsy: Localization by Pneumographic Examination of the Subdural Space. H. Olivecrona. 666.
*Vitamin Therapy of Diseases of the Neuromuscular Apparatus. A. M. Doyle and H. H. Merritt. 672.
Neuritis Associated with Acromegaly. H. W. Woltman. 680.

Case Reports:

*Presenile Disease of the Central Nervous System: Report of an Unusual Case. W. H. McMenemy and E. Pollak. 683.

Developmental Anomalies of Occipital Bone, Atlas, and Axis.—The author groups congenital malformations of the occipitocervical area into: 1. Malformation of the occipital foramen; basilar impression (platybasia). 2. Fusion of the atlas with the occiput; malformation of the atlas. 3. Fusion of other cervical vertebræ; malformation of these vertebræ. 4. Abnormal position of the axis in relation to the occiput and atlas. The associated neurological syndromes which are analysed in detail are explained either by mechanical pressure from the bony anomaly or by accompanying malformation of the central nervous system or by the combination of the two. List's paper is a most useful contribution and is well illustrated. (R.M.S.)

Convulsive Phenomena Produced by Remote Excitation.—A report based R.M.S. in a series of 125 experiments on 50 dogs on which electrical stimulation was carried out by remote excitation. (R. M. S.)

Prolonged Jugular Compression.—For diagnostic purposes the signs and symptoms of disease of the central nervous system caused by space-consuming lesions may safely be exaggerated temporarily by prolonged jugular compression. The technique of such compression by the use of the ordinary sphygmomanometer and the results obtained in 100 patients with various neurological conditions are presented. With rare exceptions, negative responses were obtained in patients with epilepsy and degenerative disease of the central nervous system. Prolonged jugular compression is thus of considerable value in differential diagnosis. For space-consuming lesions of the central nervous system and herniations of the intervertebral disc, the test proved a valuable addition to the neurological examination in corroborating the more obvious diagnoses and in contributing new data in cases in which the diagnosis was difficult and obscure. (R. M. S.)

Beta Erythroidine Hydrochloride in Hypertonia.—Clinical experience with beta erythroidine hydrochloride, a drug possessing a curare-like pharmacological action, indicates that it exerts a slight, inconstant and transient lissive (loosening) action on the skeletal musculature in hypertonic states. These effects are accompanied by such constant and undesirable side effects as to militate against its clinical use. Observations in cases of chronic encephalitis with parkinsonism indicate that the drug has no value in the treatments of tremor. Beta erythroidine hydrochloride apparently possesses a small margin of safety and is not to be recommended for clinical trial. (R. M. S.)

Polioencephalitis Hæmorrhagica of Wernicke.—Two cases of polioencephalitis hæmorrhagica associated with febrile disease are reported. The diagnosis of typhoid fever in one case was confirmed by laboratory and autopsy findings. The pathogenesis in these two cases is briefly discussed. (R. M. S.)

Corticomeningeal Scars in Traumatic Epilepsy.—Circumscribed trauma to the brain usually results in the formation of adhesions between the dura and the pia-arachoid, and pneumographic examination of the subdural space may be used to demonstrate the presence and location of scars in the brain in cases of traumatic epilepsy. A case is reported in which an epileptic syndrome in a woman of 23 could be traced to an injury to the head, though trivial at the time, received in early childhood. Pneumograms of the subdural space demonstrated the presence of a circumscribed lesion in the left frontal lobe, and the patient has now been well for 1½ years after removal of the scar in the brain. (R. M. S.)

Vitamin Therapy of Diseases of Neuromuscular Apparatus.—The authors' ex-

perience suggests that employment of vitamin therapy with muscular atrophies has been widely heralded before it has been proved to be of value. (R. M. S.)

Presenile Disease of C.N.S.—McMenemey and Pollak report a case of an unusual psychosis of rapid course in a woman aged 60. Slight degeneration of nerve cells and disproportionate hyperplasia of fibrous astroglia were found. The relation of this disease to the so-called Creutzfeldt-Jakob disease, to the parenchymatous cerebellar atrophies with dementia and to pellagra as well as to other conditions, is discussed. (R. M. S.)

BRAIN

Vol. 63. No. 4. December 1940.

*Vasomotor Reflexes in the Control of Body Temperature in Man. J. J. R. Duthie and R. M. I. Mackay. 295.
*The Spinal Complications of Paget's Disease (*Osteitis Deformans*). J. W. Aldren Turner. 321.
*Suprapituitary Adamantinoma and Eunuchism. G. M. Griffiths. 350.
*Primary Degeneration of the Granular Layer of the Cerebellum: An Unusual Form of Familial Cerebellar Atrophy Occurring in Early Life. R. M. Norman. 365.

Vasomotor Reflexes and Body Temperature.—Vasodilatation occurred in unheated limbs after another limb, the circulation of which had been occluded by pressure, was immersed in hot water. The vasodilatation was not affected by preganglionic sympathectomy or by spinothalamic tractotomy which removed afferent nervous pathways from the immersed limbs. Digital temperatures were measured by thermocouples, room temperature was controlled, but blood temperature was not read. Causes for the vasodilatation are postulated. (D. J. W.)

Spinal Complications of Paget's Disease.—Seven instances of spinal compression due to vertebral changes in Paget's disease are cited in detail. Compression may be caused by widening of the vertebral bodies, fusion lipping, or the formation of bony excrescences. Vascular changes also cause cord degeneration. (D. J. W.)

Suprapituitary Adamantinoma.—Histological study of two cases of suprapituitary cyst with lack of sexual development produced no definite evidence that the control of sexual growth is localized in the supraoptic nucleus. The lesions found were in the neurochypophysis. (D. J. W.)

Unusual Form of Cerebellar Atrophy.—The outstanding feature in two cases of familial congenital cerebellar atrophy with dementia was primary degeneration of the granular layer. The other cerebellar changes were secondary to this, and no lipoid degeneration or destruction of the basket fibres were seen. The condition therefore differs from the cerebellipetal atrophy of amaurotic family idiocy. (D. J. W.)

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Mechanism of Diuresis: Alterations in the Specific Gravity of the Blood Plasma with Onset of Diuresis in Heart Failure. H. J. Stewart. 1.
The Urinary/Fecal Coproporphyr in Ratio in Liver Disease. S. A. Localio, M. S. Schwartz, and C. F. Gannon. 7.
*Studies on the Relief of Pain by Counter-irritation. G. D. Gammon and I. Starr. 13.
A Quantitative Study of the Urinary Excretion of Hypophyseal Gonadotropin, Estrogen and Androgen of Normal Women. S. C. Werner. 21.
Effect of Varying Intake of Protein and Salts on the Composition and Specific Gravity of Urine. M. Miller, J. W. Price, and L. P. Longley. 31.
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*Measurement of the Effect on the Pain Threshold of Acetylsalicylic Acid, Acetanilid, Acetophenetidin, Aminopyrine, Ethyl Alcohol, Trichlorethylene, A Barbiturate, Quinine, Ergotamine Tartrate and Caffeine: An Analysis of Their Relation to the Pain Experience. H. G. Wolff, J. D. Hardy, and H. Goodell. 63.
Thrombocytopen: A Confirmatory Report. H. Rose and L. B. Boyer. 81.
The Measurement of Cardiac Output. An Improvement of the Acetylene Method Providing an Inherent Check. W. Adams and I. Sandiford. 87.
The Liver Lipids in Normal Human Livers and in Cases of Cirrhosis and Fatty Infiltration of the Liver. E. P. Ralli, S. H. Rubin, and S. Rinzler. 93.
Electrophoretic Analysis of the Proteins of Plasma and Serous Effusions. J. A. Luetscher. 99.

Relief of Pain by Counter-irritation.—The authors after inducing pain on themselves by the subcutaneous injection of 10 per cent. NaCl or the application of irritant ointments studied the relief afforded by counter-irritants such as heat, cold, electric current, vibration, and tactile stimulation. The application and the removal of counter-irritation both caused temporary relief. It was shown that this is not due to circulatory changes, that some of the changes could be accounted for by an altered sensory discharge, but the possibility of central depression of pain is also considered. (J. N. C.)

Effect of Drugs on Pain Threshold.—This paper is a continuation of their previous study on drugs affecting the pain threshold. Acetylsalicylic acid, acetanilid, acetophenetidin, aminopyrine, alcohol, and trichlorethylene raised the threshold, whilst caffeine sodiobenzoate, ergotamine tartrate, and quinine sulphate were without effect. (J. N. C.)

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The Renal Blood Flow in Coarctation of the Aorta. M. Friedman, A. Selzer, and H. Rosenblum. 107.
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The Toxicity of Orally Administered Potassium Salts in Renal Insufficiency. A. W. Winkler, H. E. Hoff, and P. K. Smith. 119.
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Lysolecithin and Hemolytic Anemia. The Significance of Lysolecithin Production in the Differentiation of Circulating and Stagnant Blood. K. Singer. 153.
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A Defect in the Metabolism of Tyrosine and Phenylalamine in Premature Infants. I. Identification and Assay of Intermediary Products. S. Z. Levine, E. Marples, and H. H. Gordon. 199.
A Defect in the Metabolism of Tyrosine and Phenylalamine in Premature Infants. II. Spontaneous Occurrence and Eradication by Vitamin C. S. Z. Levine, H. H. Gordon, and E. Marples. 209.
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The Cephalin-Cholesterol Flocculation Test as an Aid in the Diagnosis of Hepatic Disorders. F. J. Pohle and J. K. Stewart. 241.

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The Contemporary Setting of the Pioneer. A. Meyer. 1.
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Neurosecretion. 3. The Cerebral Organ of the Nemerteans. B. Scharrer. 109.
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Constitutional Differences Between Deteriorated and Non-Deteriorated Patients with Epilepsy: iii. Dactylographic Studies. M. Brown and H. A. Paskind. 579.
A Note on the Imagination and Its Exploitation: Psalmanazar and Hélène Smith. C. M. Campbell. 605.
A Method for Shortening the Duration of Lower Motor Neurone Paralysis by Cholinergic Facilitation. A. Wolf. 614.
Cysticercus Cyst of the Fourth Ventricle with Surgical Removal. L. J. Adelstein. 623.
Electric Changes and Corpse Reaction on the Peripheral Nerves in Insulin-Treated Schizophrenic Patients. M. Kastan. 630.
Convulsion Seizures Following Eclampsia. H. G. Hadley. 638.
The Mental and Neurological Sequellæ of Carbon Monoxide Asphyxia in a Case Observed for Fifteen Years. N. Raskin and O. C. Mullaney. 640.

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*Some Peculiar Manifestations of Memory with Special Reference to Lightning Calculators. A. A. Brill. 709.

Nasopharyngeal Tumors and Their Neurological Complications. L. A. Titrud and W. T. Peyton. 727.

Application of the Rorschach Method in an Epileptic Case with Psychoneurotic Manifestations. Z. A. Piotrowski and D. M. Kelley. 743.

The Occurrence of Complications of the Nervous System Following the Extraction of Teeth. B. J. Alpers. 752.

Compression Fractures of the Dorsal Vertebrae Resulting from a Convulsion Occurring During the Course of Insulin Shock Therapy. Report of a Case. C. Rupp. 762.

Lightning Calculators.—Most lightning calculators show their talent before they receive any education and generally lose it within a few years; however, it may be developed at a late age, as in the case of one Perini diagnosed "idiotoid." Most of the prodigies were mentally deficient and most were of the visual memory type. In none has the question of heredity been adequately investigated. But one must assume phylogenetic factors as well as the autogenetic which are often seen to hasten the development of the faculty. (W. M. H.)

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Hypoglycemia and the Autonomic Nervous System. J. D. Fortuyn. 1.

A Psychiatric Study of One Hundred Criminals J. E. Oltman and S. Friedman. 16.

*A Contribution to the "Quick-Motion-Picture" Illusion of Hoff-Pötzl. A. A. Weil. 42.

*An Analysis of Results in the Metrazol Shock Therapy of Schizophrenia. H. O. Colomb and G. L. Wadsworth. 53.

Mass-Psychosis and Its Effects. D. Abrahamsen 63.

"Quick-Motion-Picture" Illusion of Hoff-Pötzl.—The "quick-motion-picture" illusion is described in hypoglycæmia, manic excitement, and oculogyric crises. In the last the fixity of the field of vision may be a factor. In general the basic disturbance is in the incongruity of the external tempo and the inner tempo (ego-time) upon which perception depends. This disturbance is likely to occur in twilight states, (W. M. H.)

Metrazol Shock Therapy of Schizophrenia.—Ninety-seven cases of dementia præcox treated with metrazol showed a 50 per cent. increase in the recovery rate over a control group. The effect was to speed up recovery so that patients were discharged recovered rather than improved. Good results occur in cases with certain features: acute onset; duration less than one year; previous history of fair social adjustment; prepsychotic personality within average limits; prominent situational factors; affective features, confusional or deliroid states; overactivity, mutism, and negativism; rapid regression. In selecting cases for treatment content can be ignored. (W. M. H.)

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Pharmacological Shock Treatment of Involutional Melancholia. J. Solovay and F. W. Schwarz. 443.

A Modified Babinski Reflex (Resistance Reflex). E. Lichtmann. 451.

The Epileptic Insultus in a Case of Tumor of the Diencephalon. A. A. Boon and J. Doff. 453.

Progressive Muscular Atrophy and Syphilis. H. Clerckley and L. E. Geeslin. 460.

Electrokinetic Changes in the Endolymph as a Hypothetical Cause of Falling and Past Pointing due to Stimulation by Galvanic Current. L. J. Pollock, I. Finkelman, and I. C. Sherman. 473.

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The Fulfilment of White's Purpose. F. A. Fenning. 561.

Agenesis of the Corpus Callosum. L. N. Goldensohn, E. R. Clardy, and K. Levine. 567.

*Psychogenic Body Image Disturbances Associated with Psychogenic Aphesia and Agnosia. H. A. Teitelbaum. 581.

Hutchinson Teeth without Congenital Syphilis. L. Kerschbaumer. 613.

*A Comparative Study of Recovered and Deteriorated Schizophrenic Patients. O. Kant. 616.

Body Image Disturbances.—Clinical cases of hysterical body image disturbance with other signs of agnosia and aphasia may be paralleled by post-hypnotic suggestion in suitable subjects. In both groups the symptoms are similar to those due to irreversible organic brain disease. They show defects in spatial construction and

calculation such as are typically associated with disturbance of the body image. (W. M. H.)

Recovered and Deteriorated Schizophrenics.—Comparison of the clinical picture and features in groups of recovered and deteriorated schizophrenic patients shows certain features to be more frequent (ratio of 6 : 1) among the recovered—acute onset, psychogenic precipitation, clouding, extraversion, and pyknic physique. These then are fair prognostic points. Among the recovered history of gradual onset is found only in those resembling atypical depressive states. Markedly discordant behaviour and lack of clouding distinguish the deteriorated cases with depressive beginning. (W. M. H.)

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Fundamental Questions of Forced Treatment of the Mental Diseased who have Perpetrated a Crime. Z. M. Feinberg. 73.
Creative Way in the Psychiatry of J. V. Kannabich. T. I. Goldovskaya. 83.

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Observations on Cortical Somatic Sensory Mechanisms of Cat and Monkey. W. H. Marshall, C. N. Woolsey, and P. Bard. 1.
Observations on Subcortical Somatic Sensory Mechanisms of Cats under Nembutal Anesthesia. W. H. Marshall. 25.
Pupillodilator Reactions to Sciatic and Diencephalic Stimulation. A Comparative Study in Cat and Monkey. E. A. Weinstein and M. B. Bender. 44.
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Vol. 4. No. 2. March 1941.

Development of Swimming and Righting Reflexes in Frog (*Rana Guentheri*): Effects thereon of Transection of Central Nervous System before Hatching. G-H. Wang, T-W. Lu. 137.
*Thermocoagulation of Motor Cortex Exclusive of its Sixth Layer. J. P. Murphy and J. G. Dusser de Barenne. 147.
Transmission of Impulses through the Burdach Nucleus. P. O. Therman. 153.
*Influence of Discharge of Motoneurons upon Excitation of Neighboring Motoneurons. B. Renshaw. 167.
*A Direct Central Inhibitory Action of Dromically Conducted Impulses. D. P. C. Lloyd. 184.
*Effects of Lesions of Corpus Striatum upon Spontaneous Activity in the Male Rat. F. A. Beach. 191.
*Functional Anatomy of Brachium Pontis. R. S. Turner and W. H. German. 196.

Thermocoagulation of Motor Cortex.— The electrocorticogram, studied after laminar thermocoagulation of the precentral motor cortex to varying depths, was found to be significantly reduced only when the coagulation included the large and giant pyramidal cells, but not layer VI, in area 4A, and the homologous fifth layer in other precentral arm areas. Pronounced motor deficit was produced by thermocoagulation to this extent. (W. M. H.)

Influence of Motoneurone Discharge upon Excitation.—An antidromic volley in a group of motoneurones produces a small centrifugal discharge from the spinal cord into some of the motor axons which carry the antidromic impulses. These impulses seem to be repetitive discharges set up in some central portion of the motoneurones rather than reflex discharges synaptically excited. In addition, antidromic volleys may inhibit motor discharges if the tested and conditioning motor nerves are branches to the same muscle or muscle group. The response deficit arises early and is at a maximum if the volley precedes the tested motor discharges by 2 to 4 msec. Both facilitation and inhibition occur if the antidromic and tested motor impulses are in nerves to different muscles. Maximal facilitation is found when the interval is about 25 to 30 msec. (W. M. H.)

Inhibitory Action of Conducted Impulses.—In the lumbospinal cord of the cat under dial primary afferent fibres produce inhibition of motoneurons without measurable latency by primary afferent collaterals or ventrolateral column collaterals. The site of inhibition is probably the motor nucleus. (W. M. H.)

Corpus Striatum Lesions and Spontaneous Activity.—In five rats electrolytic lesions in the corpus striatum failed to alter spontaneous activity as recorded in revolving activity cages. (W. M. H.)

Brachium Pontis Anatomy.—In two mangabeys and one Java monkey, section of the brachium pontis did not affect performance in learned problems like problem boxes. Unilateral section was followed by curvature of the head and spine, spiralling and circus movements, incoordination between hind and fore limbs, hypotonia of both lower limbs for about 4 weeks. Bilateral operation had similar sequels, but in addition, progressive incoordination between the limbs in locomotion and a general sluggishness. (W. M. H.)

JOURNAL OF PHYSIOLOGY

Vol. 99. No. 1. December 1940.

The Influence of Diets Low in Magnesium upon the Chemical Composition of the Incisor Tooth of the Rat. J. Duckworth and W. Godden. 1.
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Function of Round Window.—When the round window niche in cats is occluded by plaster of Paris there is no alteration in the contralateral auditory tract response. A parallel is found in a patient whose round window was occluded by otosclerotic new bone without any impairment of hearing on that side. Thus, since free movement of the window is not essential for hearing its function cannot be adequately explained on the resonance hypothesis. It is suggested that it may act as a distensible vent in the circulation of the perilymph. (W. M. H.)

Activity of Elasmobranch Labyrinth.—Discharges from the isolated labyrinth of an elasmobranch were examined oscillographically. The horizontal canals respond to rotation about the vertical axis, the anterior and posterior vertical to rotation about all three axes. The synergy of their discharges is related to the resulting eye muscle reflexes. A silent period is shown to follow excitatory rotation and an after discharge inhibitory rotation. (W. M. H.)

Action of Venom and Lysolecithin on Adrenal Medulla.—In cats the output of adreline from the adrenals which follows injection of bee and cobra venom is attributed to the formation of lysolecithin by the strong phosphatases of the venom. In rabbits lysolecithin has an inconstant effect on the secretory activity of the adrenal medulla and causes, on injection, a fall in arterial blood pressure. (W. M. H.)

Ions and Adrenergic Transmission.—In the perfused rabbit's ear calcium is shown to decrease the action of adrenaline and to increase the response to nervous stimulation. The effects of potassium are contrary but in degree the action on the response to adrenaline is slight. (W. M. H.)

Experimental Cerebral Concussion.—Concussion was produced in cats under nembutal without any macroscopic lesion. Acceleration in movement is the essential factor for if the head is prevented from moving concussion fails to occur. Rise of intracranial pressure did not occur although it is possible to produce similar effects by a shock-like rise of intracranial pressure alone. (W. M. H.)

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Capillary Permeability and Sympathetic Nervous System.—In dogs, cats, and rabbits sympathectomy reduces the penetration of dye from the blood through the synovial membrane of the knee joint. It is suggested that a sympathetic control of permeability balances the effects of vasomotor changes. (W M. H.)

Vasoconstrictor Nerves and Oxygen Consumption in Muscle.—In the isolated perfused gastrocnemius of the dog adrenaline increases the arteriovenous temperature difference, the A—V O_2 difference and the apparent consumption of O_2; stimulation of the vasoconstrictor nerves produces a contrary result. It would appear that the latter divert blood through regions—possibly arteriovenous anastomoses—in which the O_2 consumption and surface available for heat loss are small. (W. M. H.)

Adrenaline and Nerve Action Potentials.—In cats on intra-arterial injection of adrenaline there is an increase in the height of the α β action potentials produced by submaximal stimulation of the sciatic nerve in situ. The effect is considered due to a lowering of threshold; it lags behind and outlasts the vascular effects. Adrenaline abolishes or reduces the δ spike on maximal stimuli; this effect is attributed to the anoxia of vasoconstriction. (W. M. H.)

Vaso-Dilator Action of Potassium.—If the adrenals are removed so as to prevent the release of adrenaline, the effect of small doses of KCl is vasodilatation in the hind limbs of dogs and cats. Large doses cause vasoconstriction which may be largely reduced by ergotoxine. The potentiation and depression of working muscle by KCl is not dependent upon the vascular changes but in fatigued muscle these effects may be closely related. The release of K ions may contribute to the vasodilatation in contracting muscle. (W. M. H.)

Distribution of Pituitary Antidiuretic Hormone.—Mammalian pituitary bodies contain at least eight times as much of the antidiuretic principle as the glands of any non-mammal. Thus phylogenetically the activity of the pars nervosa and the development of the Henle's loop would appear related. (W. M. H.)

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*Neuro-Muscular Transmission in the Extrinsic Muscles of the Eye. G. L. Brown and A. M. Harvey. 379.

Action of Acetylcholine, etc., on C.N.S.—Intracarotid injection of acetylcholine increases motor responses to electrical stimuli on the motor cortex of the cat. The action is antagonized by atropine and is followed by depression of responses. A/ch has similar effects intravenously but depression is prominent. Small quantities of eserine usually depress or obliterate responses to stimulation of the cortex and of the pyramidal tract. Carbon dioxide depresses cortical responses by a central action which is antagonized by atropine and potentiated by eserine. This is con-

sidered presumptive evidence of the release of A/ch by CO_2. (W. M. H.)

Skin Resistance Response.—The skin resistance response to various sensory stimuli may be elicited from any part of the limbs, body wall, and ears in the suitably warmed human subject. Its latency varies with location and the temperature of the part of the body. The response is dependent on the synchronous development of vasoconstriction and sweating. (W. M. H.)

Peripheral Conduction Rate.—In man the velocity of conduction in sympathetic nerves was determined by means of the skin resistance response. It varies from 2·17 to 1·80 m/sec. in the upper limb, from 2·30 to 2·03 m/sec. in the chest, and from 1·27 to 0·85 m/sec. in the leg. The limitations of the method are indicated. (W. M. H.)

Amine Oxadase in Sepia Officinalis.—The occurrence of an amine oxidase, showing a preference for tyramine over other sub strates, in organs of the cephalopod Sepia suggests tyramine may be a hormone in these invertebrates. No evidence is found for the presence of histamimase or a decarboxylase for tyrosine or dopa in Sepia. (W. M. H.)

Neuromuscular Transmission in Extrinsic Muscles.—In decerebrated cats the responses of extrinsic ocular muscles to nerve stimulation were found to have a refractory period of 0·5 m/sec. Facilitation of the action potential following a nerve volley, studied in preparations with curare in less than paralysing doses, reaches a peak between 0·8 and 1·5 m/sec. disappearing at a stimulus interval of 10 m/sec. to be followed by a period of depression of the second response lasting as long as 4 sec. After eserine single nerve volleys give a series of spikes undergoing a logarithmic decrement, while double nerve volleys have complex effects suggesting a prolongation of the refractory period of the muscle and interference with the second of the volleys. A/ch by artery or vein evokes a contraction with oscillatory action potential. After eserine A/ch and repetitive nerve stimulation evoke a contracture blocking propagation of excitation in the muscle fibre. (W. M. H.)

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(*a*) M. Minskowski, Präsident des Psychiatrisch-Neorologischen Vereins Zürich. (M. Minskowski, President of the Society.) 1.
(*b*) H. W. Maier; Bleulers Leben und praktisches Wirken. (Bleuler's Life and Deeds.) 10.
(*c*) E. Minskowski; Hommage à la mémoire du Professeur Eugene Bleuler. (Tribute to the memory of Professor E. Bleuler.) 15.
(*d*) L. Binswanger; Bleulers geistige Gestalt. (Bleuler's philosophy of life.) 24.
(*e*) F. Braun, im Namen des Vorstandes der Schweiz-Gesellschaft für Psychiatrie. (F. Braun, representing the Swiss Association for Psychiatry.) 29.
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